6. WAG 9 ECOLOGICAL RISK ASSESSMENT

This WAG ecological risk assessment (ERA) represents the second phase of the three phased approach to ERA (Figure 6-1) discussed in Section 6 5. The approach applies an iterative, "tiered" process in which preliminary assessments, based on conservative assumptions, support progressively more refined assessments (Maughn, 1993; Opresko et al., 1994; Levin et al., 1989).

The first phase is the screening level ERA, which is a "preassessment" or data gap analysis performed at the WAG level. The SLERA phase reduces the number of sites and contaminants to be addressed in subsequent assessments. This screening is used only as a preassessment tool to: (1) better define the extent and nature of individual WAG sites of contamination and identify sites at which no contaminants of potential concern (COPCs) are found, (2) reduce the number of COPCs to be addressed in the WAG ERA by eliminating those that clearly pose a low likelihood for risk, (3) identify sites for which further data are needed, and (4) identify other data gaps. The screening also serves to support problem formulation and determine media and pathways to be evaluated for WAG ERA assessments.

This WAG ERA is the second phase in the Idaho National Engineering and Environmental Laboratory (INEEL) Ecological Risk Assessment (ERA) process and provides a site-by-site evaluation of the risks to ecological resources as a result of exposure to radiological and nonradiological contaminants at the WAG level. The WAG 9 SLERA was conducted to screen sites identified in the Federal Facility Agreement and Consent Order (FFA/CO) (DOE-ID 1991) and to identify those contaminants present at WAG 9 that have the potential to cause undesirable ecological effects. The sites and contaminants identified as a result of the SLERA, in addition to those sites for which inadequate sampling information existed for inclusion in the SLERA are analyzed here, in the WAG ERA. This assessment was performed using the same basic methodology developed in the *Guidance Manual for Conducting Screening Level Ecological Risk Assessments at the INEEL* (VanHorn et al. 1995).

6.1 Objectives

The objectives of the ERA were as follows:

- Determine the potential for adverse effects from contaminants on ecological receptor populations and protected wildlife species (individuals and populations) at the WAG level
- Identify sites and COPCs to be assessed in the INEEL-wide ERA
- Provide input to the data gap analysis for the INEEL-wide ERA.

The INEEL approach for ERA was specifically designed to follow the direction provided by the EPA *Framework for Ecological Risk Assessment* (EPA 1992). This approach divides the ERA process into three steps: problem formulation, analysis, and risk characterization.

The goal of the problem formulation step is to investigate the interactions between the stressor characteristics, the ecosystem potentially at risk, and the ecological effects (EPA 1992). The problem formulation phase results in characterization of stressors (i.e., identification of the contaminants),

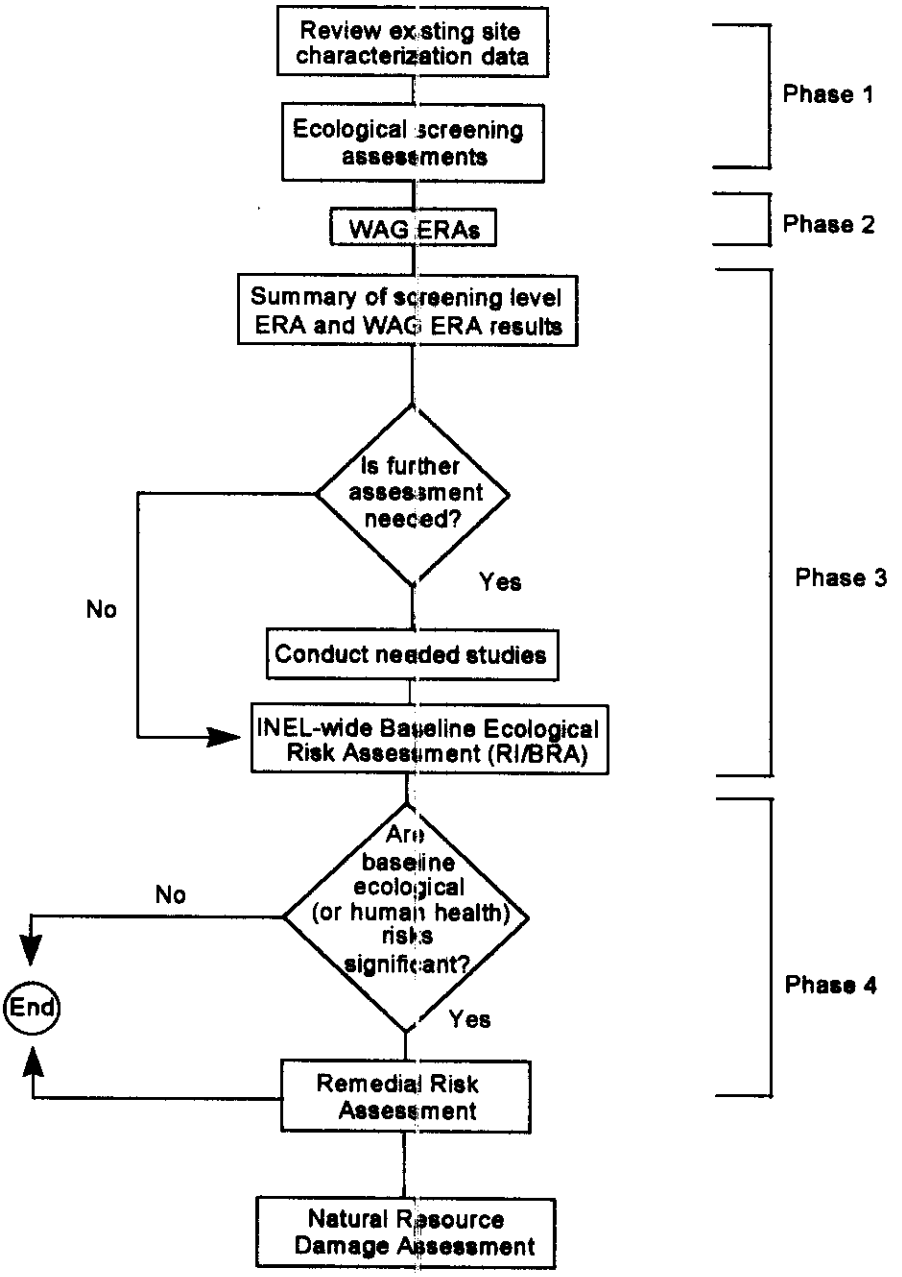

Figure 6-1. A phased approach to the INEEL ERA.

definition of assessment and measurement endpoints, and the ecological effects that will be used to analyze risk using the conceptual site model (CSM). This step of the assessment is presented in Section 6.2, WAG ERA Problem Formulation.

In the analysis step, the likelihood and significance of an adverse reaction from exposure to the stressor(s) were evaluated. The exposure assessment involves relating contaminant migration to exposure pathways for ecological receptors. The behavior and fate of the COPCs in the terrestrial environment was presented in a general manner because no formal fate and transport modeling was conducted for this WAG ERA. The ecological effects assessment consisted of hazard evaluation and dose-response assessment. The hazard evaluation involved a comprehensive review of toxicity data for contaminants to identify the nature and severity of toxic properties. Dose from multiple media (surface and subsurface soil, and surface water) identified at the INEEL were developed and used to assess potential risk to receptors. Because no dose-based toxicological criteria exist for ecological receptors, it was necessary to develop appropriate toxicity reference values (TRVs) for the contaminants and functional groups at the INEEL. A quantitative analysis was used, augmented by qualitative information and professional judgment as necessary. This step of the analysis is presented in Section 6.3, Analysis.

The risk characterization step has two primary elements (EPA 1992). The first element is the development of an indication of the likelihood of adverse effects to ecological receptors. The second element is the presentation of the assessment results in a form that serves as input to the risk management process. To determine whether there is any indication of risk due to the contaminant concentrations, exposure parameters were used to calculate dose for the key functional groups and sensitive species [threatened and/or endangered (T/E) and Category 2 (C2)]. Hazard quotients (HQs) were then calculated by dividing the calculated dose by the TRV and then used as an indicator of the potential for adverse effects. The risk characterization section of the WAG ERA is presented in Section 6.4, Risk Characterization.

6.1.1 Statutory and Regulatory Basis

The widespread application of ERAs to hazardous waste site investigations under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) began recently. In December 1988, the EPA directed that "thorough and consistent" ecological assessments should be performed at all Superfund sites (EPA 1988). This directive was based on the language in CERCLA [as amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA) and other statutes] mandating remediation of hazardous waste sites to protect the environment as well as human health. The National Contingency Plan (NCP) requires that baseline risk assessments (BRAs) characterize the current and potential threats to human health and the environment [40 CFR Part 300.430 (d)(4)], and specifies that environmental risk evaluations "assess threats to the environment, especially sensitive habitats and critical habitats of species protected under the Endangered Species Act" [40 CFR Part 300.430(e)(2)(I)(G)].

Section 121(d)(A) of CERCLA requires that Superfund remedial actions meet Federal and state standards, requirements, criteria, or limitations that "are applicable or relevant and appropriate requirements (ARARs)." ARARs are those substantive environmental protection requirements promulgated under Federal or state laws that, while not legally applicable to the circumstances at the site or facility, address situations sufficiently similar so that their use is well suited to the particular site. ARARs applicable to the WAG 9 ERA are listed in Table 6-1. A further discussion of ARARs is included in the Guidance Manual (VanHorn et al. 1995).

Table 6-1. ARARs for the WAG 9 ERA.

Requirement	Authority	Trigger
Endangered Species Act	16 USC 1531-1543	Location specific
Threatened Fish and Wildlife	50 CFR 227.4	Location specific
Migratory Bird Conservation	16 USC 715	Location specific
Migratory Bird Treaty Act	16 USC 702	Location specific
Protection of Bald and Golden Eagles Act	16 USC 1531	Location specific
Idaho Fish and Wildlife Act	16 USC 756, 757	Location specific
Wetlands Conservation Act	16 USC 4404	Location specific

Recognizing the need, the Department of Energy (DOE) published *Incorporating Ecological Risk Assessment into Remedial Investigation/Feasibility Study Work Plans* (DOE, 1994). This document "provides guidance to the U.S. Department of Energy staff and contractor personnel for incorporation of ecological information into environmental remediation planning and decision making at CERCLA sites." (DOE, 1994).

Compliance with ARARs is a threshold requirement that a remedial/restoration activity must meet to be eligible for selection as a remedy. ARARs are either chemical-, action-, or location-specific, depending upon whether the requirement is triggered by the presence or emission of a chemical, by a particular action, or by a vulnerable or protected location. A list of the definitions of these ARARs follows:

- Chemical-specific—Risk-based numerical values or methodologies that establish an acceptable amount of concentration of a contaminant in the ambient environment
- Action-specific—Technology or activity-based requirements for remedial/restoration actions
- Location-specific—Restrictions placed upon the concentration of hazardous substances or the conduct of activity at a given location.

Only location-specific ARARs are applicable in the WAG 9 ERA.

The WAG 9 ERA addresses issues related to all ARARs listed for WAG 9 in Table 6-1, except the Wetlands Conservation Act. This ARAR is included since, wetland habitat has appeared on maps as part of the Fish and Wildlife National Wetlands Inventory (Hampton et al. 1995). These are generally waste ponds that are generated solely due to facility activities and preliminary surveys indicate that most do not meet formal wetland classification criteria (ACOE 1987). However, if final evaluation indicates that these do meet formal designation criteria they will be evaluated based on ARAR considerations. T/E and/or sensitive species as protected by ARARs are discussed in Section 6.2.4.

6.2 WAG ERA Problem Formulation

The goal of the problem formulation step of the ERA is to investigate the interactions between the stressor characteristics, the ecosystem potentially at risk, and the ecological effects (EPA 1992). This

process begins with a general description of the site and previous investigations, and a characterization of the ecosystem at risk. Next, the potential stressors to the ecosystem are identified, the migration pathways of the identified stressors are modeled, and the potentially affected components of the ecosystem are identified. The ecosystem at risk and stressor characterization with exposure pathways are then assimilated into the CSM. The problem formulation step results in characterization of stressors (i.e., identification of the COPCs), definition of the assessment endpoints, and pathway/exposure models that will be used to analyze risk using the CSM. Primary elements of the problem formulation step for the WAG 9 ERA are described in the following sections.

6.2.1 Overview of WAG 9

WAG 9 includes hazardous waste release sites at the Argonne National Laboratory-West (ANL-W). The ANL-W facility is located in the extreme southeastern portion of the INEEL as shown in Figure 1-2. The Argonne Research Area (ARA) consists of seven major complexes: Experimental Breeder Reactor II (EBR-II), the Transient Reactor Test Facility (TREAT), the Zero Power Physics Reactor (ZPPR), the Hot Fuel Examination Facility (HFEF), the Fuel Cycle Facility (FCF), the Fuel Manufacturing Facility (FMF), the Laboratory and Office Building, and support complexes such as the Radioactive Liquid Waste Treatment Facility (RLWTF), the Radioactive Scrap and Waste Facility (RSWF), and the Sodium Process Facility (SPF). The primary mission of ANL-W for the first 20 years of operation was the basic support of the research and development for liquid metal-cooled reactors. The current mission is to prove an electro-refining process, which would melt down waste and prepare it for burial.

Radioactive wastes generated from ANL-W are primarily associated with irradiated experimental fuel subassemblies and capsules from EBR-II and, to a lesser degree, TREAT. After irradiation in ANL-W reactors, the subassemblies and capsules were conveyed to appropriate facilities for dismantling, sampling, and examination. If they were not contaminated with sodium (the coolant used in EBR-II), these reactor pieces and parts were shipped to the RWMC as remote-handled waste. Sodium-contaminated reactor parts were stored in the RSWF at ANL-W.

6.2.2 Sites of Concern

As discussed in the Guidance Manual (VanHorn et al. 1995) the sites identified in the FFA/CO (DOE-ID 1991) are initially eliminated from consideration in the WAG 9 ERA analysis based on whether the site is uncontaminated (no source to the environment) or because the site is inaccessible to the ecosystems of concern (no pathway to the environment). All sites at WAG 9 were reviewed for possible elimination from consideration in the WAG 9 ERA. Table 6-2 includes justification for eliminating sites from consideration.

The final list of sites to be included in the ERA analysis (sites of concern) is presented in Table 6-3. This table lists the COPCs identified at each site, and provides a brief description and size of each site. Refer to Figure 6-2 which illustrates the locations of the individual sites of potential concern to ecological receptors Argonne. More complete descriptions of the sites of concern for both human and ecological health are presented in Section 3.

6.2.3 Ecosystem Characterization

INEEL is located in a cool desert ecosystem characterized by shrub-steppe vegetative communities typical of the northern Great Basin and Columbia Plateau region. The surface of INEEL is relatively flat,

Table 6-2. WAG 9 operable unit and site descriptions.

OU	Site code	Sites description	Track	In	Justification
—	ANL-10	Dry Well between T-1 and ZPPR Mound	NFA		No hazardous constituents were disposed into this system. No source.
—	ANL-11	Waste Retention Tank 783	NFA		No hazardous constituents were disposed into the tank. No source.
—	ANL-12	Suspect Waste Retention Tank by 793	NFA		Site was decontaminated in 1979 and tank was removed. No source.
—	ANL-14	Septic Tank and Drain Fields (2) by 753	NFA		Site was decontaminated in 1979 and tank was removed. No source.
—	ANL-15	Dry Well by 768	NFA		Trace quantities of hydrazine disposed in drywell. Would have acted as an oxygen scavenger and been consumed. No source.
—	ANL-16	Dry Well by 759	NFA		Trace quantities of hydrazine disposed in drywell. Would have acted as an oxygen scavenger and been consumed. No source.
—	ANL-17	Dry Well by 720	NFA		No hazardous constituents were ever disposed into this system. No source.
—	ANL-18	Septic Tank and Drain Field by 789	NFA		This site contained sanitary wastes only. It was removed in 1979. No source.
—	ANL-20	Septic Tank and Leach Field by 793	NFA		This tank contained only sanitary wastes. No source.
—	ANL-21	TREAT Suspect Waste Tank and Leaching Field (Non-Rad)	NFA		This site was a non-radioactive collection tank, and no hazardous constituents were disposed into this system. No source.
—	ANL-22	TREAT Septic Tank and Leaching Field	NFA		No hazardous constituents were disposed into this system. No source.
—	ANL-23	TREAT Seepage Pit and Septic Tank W of 720	NFA		No hazardous constituents were disposed into this tank. It was filled with sand in 1980. No source.
—	ANL-24	Lab and Office Acid Neutralization Tank	NFA		No hazardous constituents were disposed into this tank. No source.
—	ANL-25	Interior Building Coffin Neutralization Tank	NFA		This above-ground tank was used to hold HNO_3 and NaOH for neutralization purposes. No source or pathway.
—	ANL-26	Critical Systems Maintenance Degreasing Unit	NFA		This active tank is self-contained within a building and serviced by a recycling company. There is no evidence of leakage. No pathway.
—	ANL-27	Plant Services Degreasing Unit	NFA		This active tank is self-contained within a building and serviced by a recycling company. There is no evidence of leakage. No source or pathway.
—	ANL-32	TREAT Control Building 721 Septic Tank and Leach Field	NFA		No hazardous constituents were ever disposed into this system. No source.
—	ANL-33	TREAT Control Building 721 Septic Tank and Seepage Pit (removed 1978)	NFA		No hazardous constituents were ever disposed into this system. No source.
9-01	ANL-63	Septic Tank 789-A	T1		This system received sanitary effluent only. No source.
9-01	ANL-60	Knawa Butte Debris Pile	T1		Test excavations revealed only construction debris (concrete, dirt, rock, rebar). No source.

Table 6-2. (continued).

OU	Site code	Sites description	Track	In	Justification
9-01	ANL-61	EBR-II Transformer Yard	T1		Site was cleaned. No source.
9-01	ANL-61A	PCB spill next to ANL-61		X	
9-01	ANL-62	Sodium Boiler Building (766) Hotwell	T1	X	
9-01	ANL-04	ANL Sewage Lagoons	T1	X	
9-01	ANL-19	Sludge Pit W of T-7 (Imhoff Tank)	T1		This site received sanitary wastes only. No known hazardous constituents were disposed in this tank. Tank was cut down 1 ft below grade in 1978 and filled with dirt. No source.
9-01	ANL-28	EBR-II Sump (regeneration)	T1		This sump was used to direct cooling tower blowdown regeneration effluent, ion exchange regeneration effluent, and small amounts of water laboratory chemicals. No source.
9-01	ANL-29	Industrial Waste Lift Station	T1	X	
9-01	ANL-30	Sanitary Waste Lift Station	T1		This concrete sump was 16 ft below grade. No pathway.
9-01	ANL-36	TREAT Photo Processing Discharge Ditch	T1	X	
9-02	ANL-08	EBR-II Leach Pit (radioactive)	T2		This site was excavated and concrete lid removed in 1993. Bottom was filled with bentonite and backfilled. No source. Sludges remain but below 10 feet. No pathway.
9-03	ANL-05	ANL Open Burn Pits #1, #2, #3	T2	X	
9-03	ANL-31	Industrial/Sanitary Waste Lift Station	T2		Chromium and radionuclides have been detected in one side of tank; however, site is a 6 ft × 6 ft × 14 ft deep reinforced concrete container. No pathway.
9-03	ANL-34	Fuel Oil Spill by Building 755	T2		Fifty gal of diesel fuel were spilled in early 1960s at this site. It is believed that PAHs have naturally degraded over the 30-year period since the spill. No source.
9-04	ANL-01	Industrial Waste Pond and Cooling Tower Blowdown Ditches (3)	RI/FS	X	
9-04	ANL-01A	Main Cooling Tower Blowdown Ditch Tank	RI/FS	X	
9-04	ANL-09	ANL Interceptor Canal	RI/FS	X	
9-04	ANL-35	Industrial Waste Lift Station Discharge Ditch	RI/FS	X	
9-04	ANL-53	Cooling Tower Riser Pits	RI/FS		Contamination below 10 feet. No pathway.

a Stage in CERCLA process as follows: NFA—No Further Action; initial investigation determined sites were uncontaminated and no source present. T1—Track 1; T2—Track 2; IA—Interim Action; RI—RI/FS.

b. Sites marked with "X" were not screened out in WAG 9 ERA initial site review.

Table 6-3. WAG 9 Operable units and sites of concern.

OU	Site code	Sites description	Site (m^2)	COPCs	Contaminated media	Comments
9-01	ANL-61A	PCB spill next to ANL-61	19	PCBs	Subsurface soil	PCB spill.
9-01	ANL-62	Sodium Boiler Building (766) Hotwell	675	Tritium, hydrazine	Surface and subsurface soil	Tritium levels 10^5 μCi/mL. No evidence of migration.
*9-01	ANL-04	ANL Sewage Lagoons	7,200	Metals, radionuclides	Sediment	Received sanitary waste, photoprocessing solutions, silver. 1974 leak—10^6 gallons. Now evaporative ponds.
9-01	ANL-29	Industrial Waste Lift Station	9	Silver	Surface and subsurface soil	EP Toxicity Test detected silver at < 1 ppm.
9-01	ANL-36	TREAT Photo Processing Discharge Ditch	243	Silver	Surface and subsurface soil	
*9-03	ANL-05	ANL Open Burn Pits #1, #2, #3	10,890	Metals, radionuclides, VOCs, PAHs, and dioxin/furans	Surface and subsurface soil	Pits operated from 1960–1970
*9-04	ANL-01	Industrial Waste Pond and Cooling Tower Blowdown Ditches (3)	12,140	Metals, radionuclides, VOCs, and herbicides	Surface and subsurface soil, sediment, surface water	Only chromium and lead exceed the EP Toxicity levels. Two ditches still in operation
*9-04	ANL-01A	Main Cooling Tower Blowdown Ditch	288	Metals, radionuclides, and semivolatile organic compounds	Surface and subsurface soil	Only chromium and silver exceed TCLP limits
*9-04	ANL-09	ANL Interceptor Canal	3,848	Metals and radionuclides	Surface and subsurface soil	Received ion exchange column regeneration products, except for inadvertent rad waste discharges
*9-04	ANL-35	Industrial Waste Lift Station Discharge Ditch	900	Metals, radionuclides, VOCs, and dioxin/furans	Surface and subsurface soil, surface water	Only silver exceeds EP Toxicity levels. Soil was excavated in 1988

*These sites will be included in the WAG 9 Comprehensive Remedial Investigation/Feasibility Study.

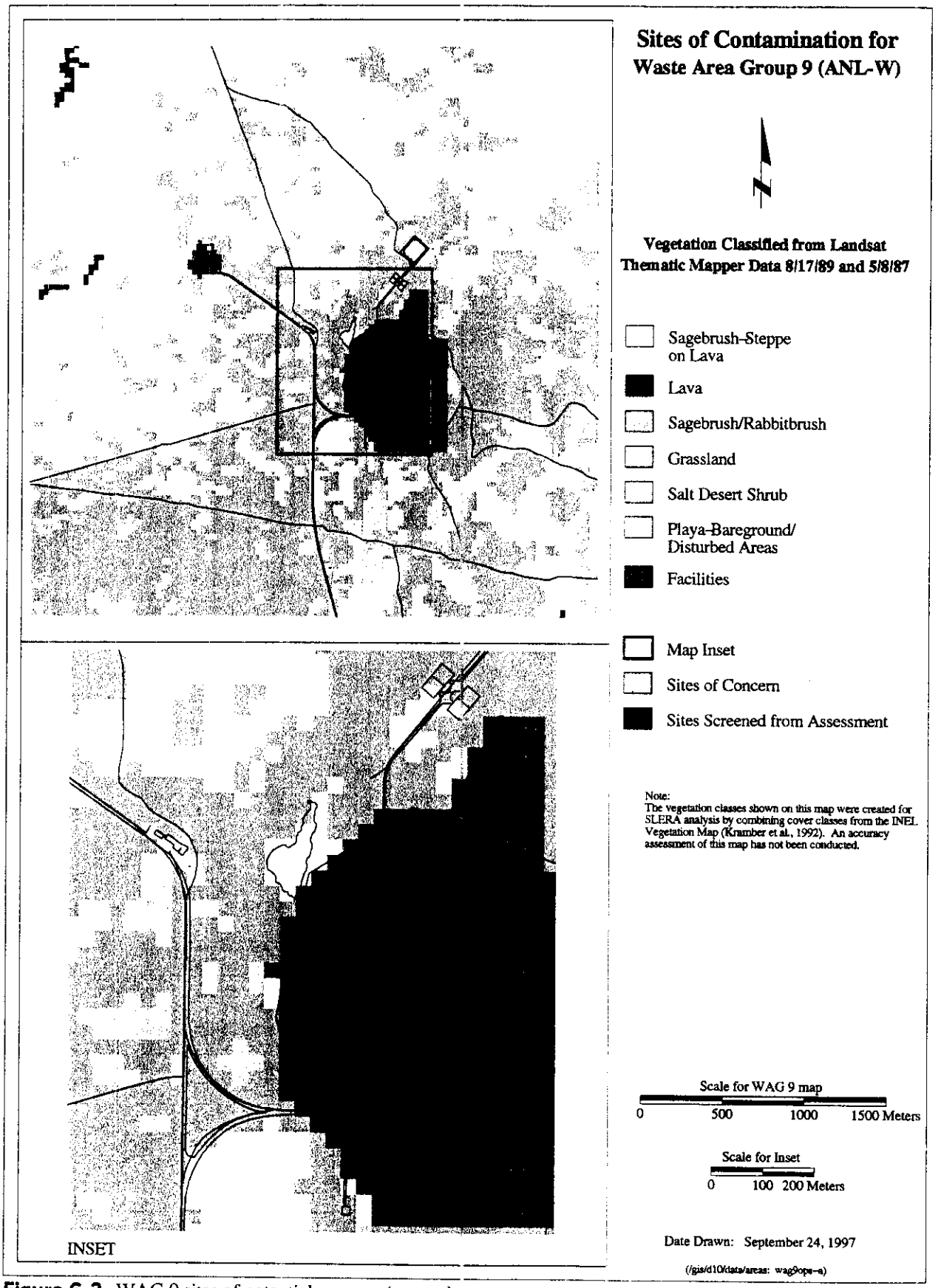

Figure 6-2. WAG 9 sites of potential concern (orange).

with several prominent volcanic buttes and numerous basalt flows that provide important habitat for small and large mammals, reptiles, and some raptors. The shrub-steppe communities are dominated by sagebrush *(Artemisia* spp.) and provide habitat for sagebrush community species, such as sage grouse *(Centrocercus urophasianus)*, pronghorn *(Antilocapra americana)*, and sage sparrows (*Amphispiza belli*). Rabbitbrush (*Chrysothamnus* spp.), grasses and forbs, salt desert shrubs *(Artiplex* spp.), and exotic/weed species comprise other communities. Juniper woodlands occur near the buttes and in the northwest portion of INEEL; these woodlands provide important habitat for raptors and large mammals. Limited riparian communities exist along intermittently flowing waters of the Big Lost River and Birch Creek drainages.

The area addressed by the WAG 9 ERA is shown on Figure 6-3 (in red). The assessment area is circular, centered between TREAT and the main facility complex, and encompasses approximately 688 hectares. The dimensions of the assessment area for WAG 9 were determined using soil contaminant sampling data and gamma detection data from aerial fly-overs (Jessmore et al. 1994). The maximum distance from WAG 9 contaminant sources for which above-background contaminant levels were detected was approximately 982 m (3,222 ft) (Jessmore et al. 1994). An ecological effects buffer of 491 m (1,611 ft) (or 1/2 the source to background distance) was added to ensure calculation of maximum exposures for species whose home ranges overlap areas of above and below background contaminant levels.

Figure 6-3 also illustrates vegetation communities and soil types associated with the assessment area. These components are discussed in detail in the following sections.

6.2.4 Biotic Components

Wildlife species present in and around ANL-W include birds, mammals, and reptiles that are associated with facilities, sagebrush-steppe habitat, rock outcroppings, deciduous trees and shrubs, and water (e.g., facility ponds and drainage areas), and both aquatic and terrestrial species are potentially present. Sagebrush-steppe habitat supports a number of species including sage grouse, pronghorn, elk (*Cervus elaphus*), and waterfowl (all important game species). Grasslands provide habitat for species such as the western meadowlark (*Sturnella neglecta*) and mule deer (also a game species). Rock outcroppings support species such as bats, woodrats, and T/E species such as the pygmy rabbit (*Brachylagus idahoensis*). Facilities within the WAG 9 assessment area also provide important habitat. Buildings, lawns, ornamental vegetation, and ponds are utilized by a number of species such as waterfowl, raptors, rabbits, and bats. No areas of critical habitat as defined in the Code of Federal Regulations (40 CFR Part 300) are known to exist within in or around ANL-W. However, the industrial waste pond located northwest of the facility supports wetland species including bulrush (*Scirpus acutus*) and cattails (*Typha latifolia*) and has been mapped as part of the Fish and Wildlife Service (FWS) National Wetlands Inventory (NWI) (Hampton et al. 1995). A number of studies have been conducted citing waterfowl and other wildlife use of this area. A lava cave is located within one mile of the assessment boundary.

The flora and fauna existing around ANL-W are representative of those found across the INEEL (Arthur et al. 1984; Reynolds et al. 1986) and are described in the following sections. Vegetation was quantitatively assessed for associations within ANL-W as part of the siting survey for the Safety Research Experiment Facilities (SREF) EIS (ERDA 1977). More recently flora in the assessment area was characterized using a vegetation map constructed for the INEEL from a combination of LANDSAT imagery and field measurements from vegetation plots (Kramber et al. 1992). Fauna potentially existing within the assessment area was identified primarily from a 1986 vertebrate survey performed on the INEEL

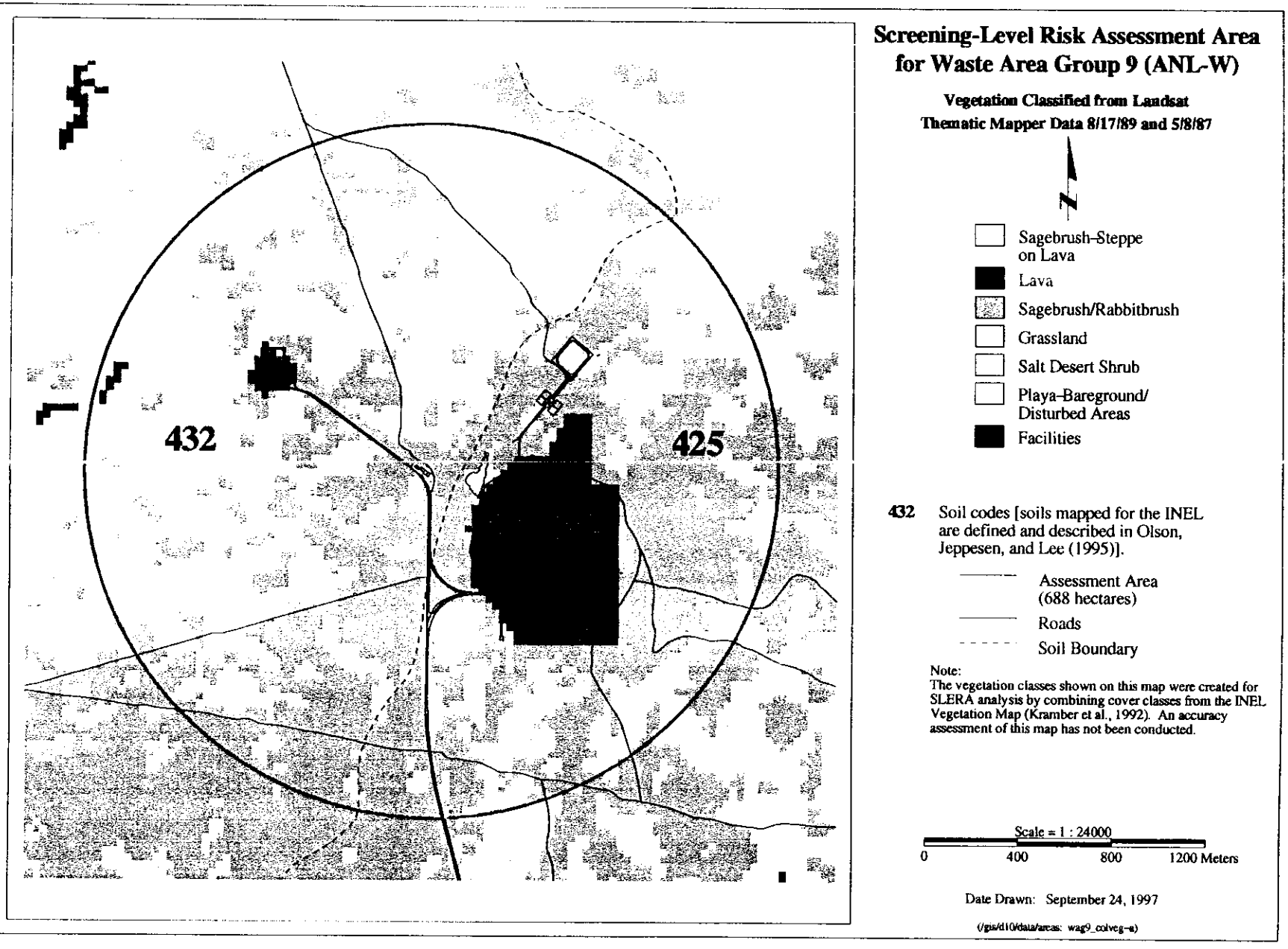

Figure 6-3. Vegetation and soil map of the WAG 9 assessment ar

(Reynolds et al. 1986) and from data collected as part of the 1977 EIS for the SREF (ERDA 1977). Information presented here is supported by previous field surveys and observations previously described in Appendix E.

6.2.4.1 Flora. The 15 INEEL vegetation cover classes defined using Landsat imagery data (Kramber et al. 1992) have been combined into eight cover classes for the WAGs (VanHorn et al. 1995). The vegetation surrounding ANL-W is shown in Figure 6-3 represents five vegetation cover classes, including sagebrush-steppe on lava, sagebrush/rabbitbrush, grassland, salt desert shrub, and playa-bareground/disturbed. The species composition for each of these classes is summarized on Table 6-4. Sagebrush/rabbitbrush and sagebrush-steppe on lava are the predominant vegetation type comprising approximately 86% of the vegetation in the assessment area. The dominant vegetation species within this community are Wyoming big sagebrush (*Artemisia tridentata spp. wyomingensis*) and green rabbitbrush (*Chrysothamnus viscidiflorus*). Grasslands present in the assessment area are comprised primarily of

Table 6-4. Vegetation cover class summary for WAG 9 assessment area.

SLERA vegetation cover class	INEEL Vegetation Cover Classes	Dominant Species
Grasslands	Steppe Basin Wildrye Grassland	*Leymus cinereus* *Descurainia sophia* *Sisymbrium altissimum* *Agropyron dasytachyum* *Artemisia tridentata* ssp. *wyomingensis* *Elymus elymoides* *Chrysothamnus viscidiflorus*
Sagebrush/rabbitbrush	Sagebrush-steppe off lava Sagebrush-winterfat Sagebrush-rabbitbrush	*Artemisia tridentata* ssp. *wyomingensis* *Chrysothamnus viscidiflorus* *Bromus tectorum* *Sisymbrium altissimum* *Oryzopsis hymenoides*
Salt desert shrubs	Salt Desert Shrub	*Atriplex nuttallii* *Atriplex canescens* *Atriplex confertifolia* *Ceratoides lanata*
Sagebrush-steppe on lava	Sagebrush-steppe on lava	*Artemisia tridentata* ssp. *wyomingensis* *Oryzopsis hymenoides* *Chrysothamnus viscidiflorus*
Playa-bareground/disturbed areas	Playa-bareground/gravel borrow pits old fields, disturbed areas, seedings	*Kochia scoparia* *Salsola kali* *Artemisia tridentata* ssp. *wyomingensis* *Chrysothamnus viscidiflorus*

wheat grasses (Agropyron spp.). Wetland species are supported by intermittent standing water from facility drainage and disposal ponds. Playa-bareground/disturbed areas cover classes represent approximately 3% of the WAG 9 assessment area vegetation cover. drainages.

6.2.4.2 Fauna. A comprehensive list of fauna potentially present at and surrounding ANL-W is presented in Appendix F. The list incorporates the concept of functional grouping as described in detail in Appendix E (Hampton and Morris 1995) of the Guidance Manual (VanHorn et al. 1995). The functional grouping approach is designed to group similar species to aid in analyzing the effects of stressors on INEEL ecosystem components. The primary purpose for functional grouping is to apply existing data from one or more species within the group to assess the risk to the group as a whole. Functional groups are used to perform a limited evaluation of exposures for all potential receptors and provide a mechanism for focusing subsequent analyses on receptors that best characterize potential contaminant effects.

Functional groups designed to be representative of receptors at WAG 9 have been identified from those listed in Appendix F. The functional groups evaluated in the WAG 9 ERA were selected with the assumption that those groups would be conservative indicators of effect for other similar groups. Species characteristics including trophic level, breeding, and feeding locations were used to construct functional groups for INEEL species. Individual groups were assigned a unique identifier consisting of a one- or two-letter code to indicate taxon (A = amphibians, AV = birds, M = mammals, R = reptiles, I = insects), and a three-digit code derived from the combination of trophic category and feeding habitats. For example, AV122 represents the group of seed-eating (herbivorous) bird species whose feeding habitat is the terrestrial surface and/or understory. The trophic categories (first digit in three-digit code) are as follows: 1 = herbivore, 2 = insectivore, 3 = carnivore, 4 = omnivore, and 5 = detrivore. The feeding habitat codes (second and third digits in three-digit code) are derived as follows:

- 1.0 Air
- 2.0 Terrestrial
  - 2.1 Vegetation canopy
  - 2.2 Surface/understory
  - 2.3 Subsurface
  - 2.4 Vertical habitat (man-made structures, cliffs, etc.)
- 3.0 Terrestrial/Aquatic Interface
  - 3.1 Vegetation canopy
  - 3.2 Surface/understory
  - 3.3 Subsurface
  - 3.4 Vertical habitat
- 4.0 Aquatic
  - 4.1 Surface water
  - 4.2 Water column
  - 4.3 Bottom

Species potentially present at and surrounding WAG 9 represent all 23 INEEL avian functional groups and nine of 10 mammalian functional groups. Both reptilian functional groups are represented by species inhabiting the immediate area. Amphibians are likely to be present at the industrial waste pond but

no formal surveys have been conducted to document their presence. No surface hydrology exists to support fish. Aquatic invertebrates, however, are supported by habitat provided by facility disposal and drainage ponds (Cieminski 1993).

Both aquatic and terrestrial invertebrates and microorganisms are present at ANL-W. Invertebrates are important links in dietary exposure for wildlife, and also may function as a good indicator for contaminant exposure in soil, aquatic systems, and vegetation uptake, and microorganisms play an important role in ecosystem processes. A list of terrestrial invertebrates present in and surrounding ANL-W can be found in ERDA 1977. These ecosystem components will not be assessed in this WAG ERA.

Insects and burrowing animals have the potential for bringing subsurface soils from buried waste to the surface. No site-specific or other data were researched to confirm or evaluate this source of surface contamination, which is considered a data gap. A thorough literature analysis of this potential contamination exposure should be evaluated in the INEEL-wide ERA.

Although some population studies have been conducted for cyclic rabbit and rodent populations, several game species (e.g., pronghorn, sagegrouse) and raptors, no recent comprehensive studies have been conducted to assess either WAG-specific or INEEL-wide wildlife population status and trends with respect to contaminant effects.

Wildlife species present in and around ANL-W include birds, mammals and reptiles that are associated with facilities, sagebrush-steppe, rock outcroppings, deciduous trees and shrubs, grasslands, and water (e.g., facility ponds and drainage areas). The varying behaviors of these species include, but are not limited to, grazing and browsing on vegetation, burrowing and flying, and preying on insects and small mammals. Both aquatic and terrestrial species are potentially present. The complexity of these behaviors is significant when considering fate and transport of contaminants and the possibility of exposure to contaminants. Subsurface contamination can become surface contamination when translocated by burrowing animals, or can be introduced into the food web when plants uptake contamination and are then ingested by an herbivore. If prey, such as a small mammal, becomes contaminated by ingesting contaminated soil or vegetation, and is then captured by a predator, such as a ferruginous hawk, the contamination can be taken offsite when the hawk returns to its nest to feed nestlings. Scenarios for potential exposure of fauna to WAG 9 contaminants are discussed in Section 6.3.

The flora and fauna present in and around ANL-W are combined into a simplified food web model. Variability in environmental conditions, such as population sizes or seasons, is not considered in this model, and a constant environment is assumed. Because both aquatic and terrestrial habitats are present at ANL-W, the model incorporates both terrestrial and aquatic species, including the decomposers, producers (vegetation), primary consumers or herbivores (e.g., rodents), secondary consumers or carnivores (e.g., snakes), and tertiary or top carnivores (e.g., raptors). The dietary relationships between each level were incorporated to identify direct and indirect exposure to contaminants for the conceptual site model as discussed later in this section

6.2.4.3 Threatened, Endangered, or Sensitive Species. No threatened, endangered (T/E), or sensitive plant species known to exist within one mile of the WAG 9 assessment area boundary.

T/E, or sensitive species that have potential for utilizing habitats in and surrounding ANL-W are listed in Table 6-5. C2 species including the burrowing owl (*Athene cunicularia*), the loggerhead shrike (*Lanius ludovicanus*), the white-faced ibis (*Plegadis chihi*), pygmy rabbit (*Brachylagus idahoensis*) and

Table 6-5. Threatened and endangered species, special species of concern, and sensitive species that may be found on the INEEL.[a] Species in **bold** are those TE and Category 2 (C2) species included for WAG 9 screening.

Common names	Scientific Name	Federal Status[b,c]	State Status[c]	BLM Status[c]	USFS[f] Status[c]	INPS Status[c]
Plants						
Lemhi milkvetch	*Astragalus aquilonius*	—	—	S	S	S
Painted milkvetch[e]	*Astragalus ceramicus var. apus*	3c	—	—	—	M
Plains milkvetch	*Astragalus gilviflorus*	NL	—	S	S	1
Winged-seed evening primrose	*Camissonia pterosperma*	NL	—	S	—	S
Nipple cactus	*Coryphantha missouriensis*	NL	—	—	—	R
Spreading gilia	*Ipomopsis (Gilia) polycladon*	NL	—	S	—	2
King's bladderpod	*Lesquerella kingii var. cobrensis*	—	—	—	—	M
Tree-like oxytheca[e]	*Oxytheca dendroidea*	NL	—	R	—	R
Inconspicuous phacelia[d]	*Phacelia inconspicua*	C2	SSC	S	S	
Puzzling Halimolobos	*Halimolobos perplexa var. perplexa*	—	—	—	S	M
Birds						
Peregrine falcon	***Falco peregrinus***	**LE**	**E**	—	—	
Merlin	*Falco columbarius*	NL	—	S	—	
Gyrfalcon	*Falco rusticolus*	NL	SSC	S	—	
Bald eagle	***Haliaeetus leucocphalus***	**LT**	**T**	—	—	
Ferruginous hawk	***Buteo regalis***	**C2**	**SSC**	**S**	—	
Black Tern	***Chlidonias niger***	**C2**	—	—	—	
Northern pygmy owl[d]	*Glaucidium gnoma*	—	SSC	—	—	
Burrowing owl	***Athene cunicularia***	**C2**	—	**S**	—	
Common loon	*Gavia immer*	—	SSC	—	—	
American white pelican	*Pelicanus erythrorhynchos*	—	SSC	—	—	
Great egret	*Casmerodius albus*	—	SSC	—	—	

Table 6-5. (continued).

Common names	Scientific Name	Federal Status[b,c]	State Status[c]	BLM Status[c]	USFS[f] Status[c]	INPS Status[c]
White-faced Ibis	***Plegadis chihi***	**C2**	—	—	—	
Long-billed curlew	*Numenius americanus*	3c	—	S	—	
Loggerhead shrike	***Lanius ludovicianus***	**C2**	**NL**	**S**	—	
Northern goshawk	***Accipiter gentilis***	**C2**	**S**	—	**S**	
Swainson's hawk	*Buteo swainsoni*	—	—	S	—	
Trumpeter Swan	***Cygnus buccinator***	**C2**	**SSC**	**S**	**S**	
Sharptailed grouse	*Tympanuchus phasianellus*	C2	—	S	S	
Boreal owl	*Aegolius funereus*	—	SSC	S	S	
Flammulated owl	*Otus flammeolus*	—	SSC	—	S	
Mammals						
Pygmy rabbit	***Brachylagus (Sylvilagus) idahoensis***	**C2**	**SSC**	**S**	—	
Townsend's western big-eared bat	***Plecotus townsendii***	**C2**	**SSC**	**S**	**S**	
Merriam's shrew	*Sorex merriami*	—	S	—	—	
Long-eared myotis	*Myotis evotis*	C2	—	—	—	
Small-footed myotis	***Myotis subulatus***	**C2**	—	—	—	
Western pipistrelle[d]	*Pipistrellus hesperus*	NL	SSC	—	—	
Fringed myotis[d]	*Myotis thysanodes*	—	SSC	—	—	
California Myotis[d]	*Myotis californicus*	—	SSC	—	—	
Reptiles and Amphibians						
Northern sagebrush lizard	***Sceloporus graciosus***	**C2**	—	—	—	
Ringneck snake[d]	*Diadophis punctatus*	C2	SSC	S	—	
Night snake[e]	*Hypsiglena torquata*	—	—	R	—	
Insects						
Idaho pointheaded grasshopper[d]	*Acrolophitus punchellus*	C2	SSC	—	—	

Table 6-5. (continued).

Common names	Scientific Name	Federal Status[b,c]	State Status[c]	BLM Status[c]	USFS[f] Status[c]	INPS Status[c]
Shorthead sculpin[d]	*Cottus confusus*	—	SSC	—	—	

a. This list was compiled from the U.S. Fish and Wildlife Service (USFWS) (letter dated April 30, 1996) the Idaho Department of Fish and Game Conservation Data Center threatened, endangered, and sensitive species for the State of Idaho (CDC 1994) and Radiological and Environmental Sciences Laboratory (RESL) documentation for the INEEL (Reynolds 1994; Reynolds et al. 1986).

b. The USFWS no longer maintains a candidate (C2) species listing but addresses former listed species as "species of concern" (USFWS April 30, 1996). The C2 designation is retained here to maintain consistency with previous assessments.

c. Status Codes: S=sensitive; 2=State Priority 2; 3c=no longer considered for listing; M=State monitor species; NL=not listed; 1=State Priority 1; LE=listed endangered; LT=listed threatened; E=endangered; SSC=species of special concern; and C2=Category 2 (defined in CDC 1994). BLM=Bureau of Land Management; INPS=Idaho Native Plant Society; R=removed from sensitive list (non-agency code added here for clarification).

d. No documented sightings at the INEEL, however, the ranges of these species overlap the INEEL and are included as possibilities to be considered for field surveys.

e. Recent updates resulting from Idaho State Sensitive Species meeting [BLM, USFWS, INPS, United States Forest Service (USFS)] - (INPS 1995, 1996)

f. USFS Region 4.

sagebrush lizard (*Sceloporus graciosus*) have been recorded in the assessment area. The presence/status of Townsend's western big-eared bat (*Plecotus townsendii*), long-eared myotis (*Myotis evotus*) and the small-footed myotis (*Myotis subulatus*) is unknown; however, the proximity to suitable roosts (caves) and open water (industrial waste pond) provide reason to expect these species could inhabit the assessment area. Other sensitive bird species include the northern harrier (*Circus cyaneus*), the red-tailed hawk (*B. jamaicensis*), the prairie falcon (*F. mexicanus*), the american kestrel (*Falco sparverius*), short-eared owl (*A. flammeus*), have also been observed inside the assessment area. Sage grouse breeding grounds (leks) are found both inside the assessment area and within one mile of the assessment boundary. The sagebrush lizard (*Sceloporous graciosus*) is the only sensitive reptile species with a potential presence at WAG 9. All of these species are State or Federal species of special concern (formerly C2). No critical habitat is known to exist in the WAG 9 assessment area. Because potential risks associated with contaminant exposures for T/E and species of special concern are of interest for both individuals and populations, those species most likely to contact WAG 9 sites and contaminants of concern have been evaluated for individual exposures. Other species considered very rare INEEL-wide (see Table F-2) and considered unlikely to receive chronic doses through frequenting WAG 9 and surrounding areas are represented through evaluation of the functional group with which they are associated.

6.2.5 Abiotic Components

The ANL-W is located on the alluvial plain of the Big Lost River, in the southeastern section of the INEEL. The topography of the area is relatively flat and the predominant soils include the Malm-Bondfarm-Matheson complex (432) soils and the Coffee/Nargon/Atom complex (425) soils (see Figure 6-3).

The Malm-Bondfarm-Matheson complex (432) consists of moderately deep, well-drained, sandy-loam soils on basalt plains. A calcic horizon is present at approximately 30 cm (12 in.). Permeability of these soils is moderately rapid, and the erosion hazards for these soils are slight to moderate (Olson et al. 1995).

The Coffee/Nargon/Atom complex (425) varies from moderately deep to very deep, well drained, silty clay loam on lava plains. The permeability of these soils is moderate to moderately slow and the runoff varies from slow, for the Atom soil, to medium to rapid, for the Coffee and Nargon soils. The erosion hazard for these soils is slight to moderate (Olson et al. 1995). Several soils were also characterized as part of the SREF EIS.

Root uptake of contaminants is a complex process that depends on various soil properties such as pH, cation-exchange capacity, and organic matter content. In addition, the process is highly variable from one plant species to another. While soil-plant relationships are not specifically considered as part of the WAG 9 ERA, this information is presented to support possible comprehensive analyses.

The climate at the WAG 9 area cannot be differentiated from that of the entire INEEL because meteorological data that are ultimately reported are collected in only two locations at the INEEL. Data reported here are collected at the Central Facilities Area (CFA) National Oceanic and Atmospheric Administration meteorological station and are extrapolated to the ANL-W facility (WAG 9). The average annual temperature is 5.4°C (41.7°F) with a mean annual precipitation of 8.74 in. Annual snowfall ranges from a low of about 30 cm (12 in.) to a high of about 102 cm (40 in.) and an average of 66 cm (26 in.). Wind patterns at the assessment area are from the west-southwest or southwest approximately 40% of the time, and the average speed is 9.3 mph at 20 ft. Wind direction the remaining 60% of the time is a combination of directions, predominantly due west or northeast.

Besides the waste ponds and facility drainages at WAG 9, no other surface hydrology is present in the assessment area. Groundwater is present; however, for this assessment, it is assumed that no pathways to surface ecological receptors exist for these sites.

6.2.6 Stressor Identification and Characterization

DOE Guidance (DOE 1993) defines a stressor as "any physical, chemical, or biological entity that can induce an adverse response." CERCLA is primarily concerned with the effects of chemical stressors. At WAG 9 chemical stressors include a variety of radionuclides, organics, and metals identified at multiple sites.

6.2.6.1 Preliminary Summary of Available Sites and Data. Sites and contaminants to be considered in the WAG 9 ERA were identified by the WAG 9 SLERA. Sites of concern identified in the SLERA were reviewed and evaluated for inclusion in the WAG 9 ERA. Additional release sites identified were also evaluated. In this section, the characterization of the contaminant concentrations at the sites of concern is discussed. The primary source of data for the WAG 9 ERA is the same as that for the human health risk assessment (Section 5 contains more information on the summarization and calculation of the final data concentrations). Track 1 and Track 2 documents were also used as sources of data when data were not available from the human health risk assessment. Table 6-6 identifies the sites and contaminants evaluated in this WAG 9 ERA and whether human health data was available.

6.2.6.2 Human Health Concentration Data. Whenever possible, data from the human health risk assessment were used. The sites and contaminants for which human health concentration data were used

Table 6-6. Sites considered in the ERA.

Operable unit	Site	Contaminated Media	Human Health Data	Potential Contaminants
9-01	ANL-61A	Subsurface soil	Yes	PCBs
	ANL-62	Surface and subsurface soil	No	Tritium, hydrazine
	ANL-04	Surface-sediment	No	Radionuclides, metals
	ANL-29	Surface and subsurface soil	No	Silver
	ANL-36	Surface and subsurface soil	No	Silver
9-03	ANL-05	Surface and subsurface soil	No	Radionuclides, metals, VOCs, PAHs, dioxins/furans
9-04	ANL-01	Surface and subsurface soil, surface-sediment, surface water	Yes	Radionuclides, metals, VOCs, herbicides
	ANL-01A	Surface and subsurface soil	Yes	Radionuclides, metals, SVOCs
	ANL-09	Surface and subsurface soil	Yes	Radionuclides, metals
	ANL-35	Surface and subsurface soil, surface water	Yes	Radionuclides, metals, VOCs, dioxins/furans

are identified in Table 6-6. In soils, the 95% upper confidence level (UCL) of the arithmetic mean was used to estimate exposure-point concentrations. Maximum concentrations were used when the 95% UCL exceeded the maximum value or when the 95% UCL could not be calculated because data from only three or less samples were available. The were broken into 0 to 0.15 m (0 to 0.5 ft), 0 to 1.22 m (0 to 4 ft), and 0 to 3 m (0 to 10 ft) average concentrations. For the WAG 9 ERA, the 0.15 m (0.5 ft) concentrations were used to characterize surficial soil concentrations. The subsurface concentrations, considered to be 0.15 to 3 m (0.5 to 10 ft), are based on the 0 to 3 m (0 to 10 ft) concentrations.

6.2.6.3 Sites Not Addressed in the Human Health BRA. Five of the sites included in the WAG-9 ERA were screened from the human health risk assessment. The five sites are ANL-04 (ANL sewage lagoons), ANL-05 (ANL open burn pits #1, #2, and #3), ANL-29 (industrial waste lift station), ANL-36 (TREAT photo processing discharge ditch), and ANL-62 (sodium boiler building 766 hotwell).

6.2.6.4 New Sites. No new sites were added to the WAG 9 ERA that had not been considered in the WAG 9 screening.

6.2.6.5 Initial Screening of Sites and Contaminants. Since the initial screening of contaminants at WAG 9 in the SLERA, additional data from newly identified sites and new data from previously

identified sites became available. It is the intent of this section to provide a new screening of the sites and contaminants identified in Table 6-6 against both background concentrations and EBSLs. The background concentrations come from the INEEL Background Guidance Document (Rood et al. 1995). EBSLs were calculated specifically for the INEEL as discussed in the Guidance Manual (VanHorn et al. 1995). EBSLs are defined as concentrations of COPCs in soil (or other media) that are expected to produce any adverse effects to selected ecological receptors under chronic exposure conditions.

Tables 6-7 through 6-9 compare site concentrations to the EBSL and background values for radionuclides, organics, and inorganics, respectively. The tabled concentrations are the concentrations used in the BRA analysis when available, otherwise they are maximum observed concentrations. A total of 10 sites have potential for posing risk to WAG 9 ecological components. There were 5 sites of the 10 that had human health sampling data Blank cells in Tables 6-7 through 6-9 indicate that the contaminant was not observed at the site. If the given concentration is in a bold font, then the corresponding site and contaminant combination will be included in the WAG 9 ERA analysis.

The stepwise decision process for inclusion of a site and contaminant combination in a WAG ERA is as follows:

1. If the site concentration of the contaminant does not exceed the 95/95% upper tolerance limit (UTL) for background concentrations, then the contaminant will not be considered in the WAG ERA for that site.

2. If the site concentration of the contaminant does not exceed the EBSL concentration, then the contaminant will not be considered in the WAG ERA for that site.

3. Otherwise, the contaminant is included in the WAG ERA for the site.

Soil-Tables 6-7, 6-8, and 6-9 compare the contaminant concentrations detected in soil at sites of concern, background, and EBSLs. This screening eliminated nineteen organic contaminants, two inorganic contaminants, and all nine of the radionuclides. This resulted in one site (ANL-62) being eliminated from the assessment.

Surface Water and Sediment—The surface water and sediment screening was performed in the WAG 9 SLERA. No further screening on these contaminants will be done, and therefore, all contaminants identified in the SLERA, will be retained for analysis. Table 6-10 shows the COPCs retained as a result of the earlier screening.

6.2.7 Pathways of Contaminant Migration and Exposure

A total of 9 sites of concern have the potential for posing risk to WAG 9 ecological components through three primary media: contaminated surface soil, contaminated subsurface soil, and contaminated surface water as discussed in the following sections. These sites are listed by medium and only surface and subsurface soils are presented. There are no surface water samples analyzed for the WAG 9 assessment area; only the sediment from the sewage lagoons (ANL-04) was assessed. Contaminated perched water and groundwater sites are also present, but for this assessment, it is assumed that no pathways to surface ecological receptors exist for these sites at WAG 9. Groundwater is generally considered inaccessible to ecological receptors because of the depth to the aquifer at INEEL [200–600 ft (60–180 m)] and large

Table 6-7. Results of radionuclide screening.[a]

Radionuclide	Min EBSL (pCi/g) Background (pCi/g)	ANL-01[b]	ANL-01A[b]	ANL-04[c]	ANL-05[b]	ANL-09[b]	ANL-29	ANL-35[b]	ANL-36	ANL-61A[b]	ANL-62
Am-241	3.55E+02 1.90E-02					3.4E-02					
Cm-244	3.36E+02 NA[d]	1.1E-01				6.0E-02					
Co-60	1.18E+03 NA	1.2E-01				1.9E-01		3.7E-02			
Cs-134	1.90E+03 NA					2.0E-02					
Cs-137	4.95E+03 1.28E+00	2.9E+01	2.3E-01	3.2E-01	3.4E-01	3.1E+01		2.0E+00			
Pu-239/240	3.79E+02 1.90E-01	1.1E-01									
Sr-90	3.34+03 7.6E-01	4.5E+00		4.2E+00		5.8E+00					
Th-230	4.18E+02 1.88E+00	1.8E+00				1.8E+00					
U-238	4.64E+02 1.85E+00		2.7E+00		1.6E+00	2.3E+00		2.3E+00			

a. The soil concentrations used for screening are the highest values detected in either the surface or subsurface samples. All values in pCi/g.

b. This site was included in the human health risk assessment, therefore, the concentration used is the 95% UCL.

c. All radionuclides observed at ANL-04 will be retained in the WAG 9 ERA as part of the water ingestion pathway. ANL-04 concentrations in a bold typeface are also assessed as part of the food and soil ingestion pathways.

d. NA--No background concentration available.

Table 6-8. Results of organics screening.[a]

Contaminant	Min EBSL (mg/kg)	ANL-01[b]	ANL-01A[b]	ANL-04[c]	ANL-05[b]	ANL-09[b]	ANL-29	ANL-35[b]	ANL-36	ANL-61A[b]	ANL-62
1,1,1-Trichloroethane	4.03E+02	5.6E-02			1.3E-02			5.3E-01			
2,4-D	2.67E-01	8.7E-02	6.4E-03					9.1E-02			
2-Butanone	1.91E+01	2.0E-01			1.6E-01			1.8E-01			
Acetone	2.78E-01	1.3E-01	4.6E-02		5.7E-02			2.2E-02			
Benzo(a)pyrene	3.34E-02				5.7E-03						
bis(2-Ethylhexyl)phthalate	2.63E+00	1.2E-01	9.6E-02					3.7E-01			
Butylbenzylphthalate	1.47E+01							7.4E-02			
Chloroform	1.33E+00	1.0E-02			3.0E-03			6.1E-03			
Di-n-butylphthalate	1.54E+01	4.1E-01	1.2E-01					2.3E-01			
Di-n-octylphthalate	4.86E+01		4.8E-02					4.9E-01			
Fluoranthene	1.74E+0				8.0E-03						
HpCDD	1.10E-06[d]				1.0E-07			**4.0E-06**			
HpCDF	1.10E-06[d]							4.7E-07			
HxCDF	1.10E-06[d]							2.9E-07			
Hydrazine	7.17E-04										3.0E-03
Methylene Chloride	4.27E-01	3.0E-01	3.7E-2		3.1E-01						
OCDD	1.10E-06[d]				9.7E-07			**1.0E-05**			
OCDF	1.10E-06[d]							5.8E-07			
PCBs (Aroclor-1260)	8.20E+00									**5.5E+01**	
PeCDD	1.10E-06[d]							**2.2E-06**			
TCDF	1.10E-06[d]							4.7E-07			
Toluene	3.03E+01	2.9E-03			3.0E-03						

a. The soil concentrations used for screening are the highest values detected in either the surface or subsurface samples. All values in mg/kg.

b. This site was included in the human health risk assessment, therefore, the concentration used is the 95% UCL.

c. All organics observed at ANL-04 will be retained in the WAG 9 ERA as part of the water ingestion pathway. ANL-04 concentrations in a bold typeface are also assessed as part of the food and soil ingestion pathways.

d. Dioxins/furans.

Table 6-9. Results of inorganics screening.[a]

Contaminant	Min EBSL (mg/kg) Background (mg/kg)	ANL-01[b]	ANL-01A[b]	ANL-04[c]	ANL-05[b]	ANL-09[b]	ANL-29	ANL-35[b]	ANL-36	ANL-61A[b]	ANL-62
Aluminum	4.27E+00 2.40E+04	**3.2E+04**	1.7E+04	1.2E+04	1.3E+04	1.9E+04		**3.0E+04**			
Antimony	7.67E-01 7.40E+00	**7.7E+00**	**5.1E+01**								
Arsenic	9.01E-01 7.40E+00	**2.5E+01**	**3.5E+01**	**1.0E+01**		**9.7E+00**		**1.2E+01**			
Barium	1.08E-01 4.40E+02	**1.7E+03**	**1.0E+03**	**5.6E+02**	2.2E+02	3.4E+02		**6.5E+02**			
Beryllium	7.34E-01 3.00E+00	**3.9E+00**	**4.2E+00**	1.8E+00	1.6E+00	1.3E+00		**5.8E+00**			
Cadmium	2.63E-03 3.70E+00	**4.2E+00**	3.3E+00		4.2E-01	1.6E+00		**4.8E+00**			
Calcium	NC[d] 3.90E+04	**1.9E+05**	**9.1E+04**	**7.7E+04**	**8.0E+04**	**7.1E+04**		**1.0E+05**			
Chloride	9.81E+00 NA[e]	**4.1E+01**						**1.4E+01**			
Chromium III	3.25E+00 5.00E+01	**1.0E+04**	**7.1E+02**	**6.9E+01**	1.9E+01	2.7E+01		**5.1E+01**			
Chromium VI	1.67E-01 5.00E+01	**1.1E+03**	**7.9E+01**	7.6E+00	2.1E+00	3.0E+00		5.6E+00			
Cobalt	4.67E-01 1.80E+01	1.8E+01	**2.6E+01**	8.6E+00	7.1E+00	1.5E+01		9.2E+00			
Copper	2.17E+00 3.20E+01	**2.0E+02**	**2.9E+02**	**3.5E+02**	2.0E+01	**3.5E+01**		**1.3E+02**			
Cyanide	2.15E-02 NA	**5.9E+00**	**2.8E+00**			-		**3.9E+00**			
Fluoride	3.11E+00 NA	**9.1E+00**				-		**4.8E+00**			
Iron	NC 3.50E+04	**4.3E+04**	**4.6E+04**	1.5E+04	1.5E+04	2.6E+04		**5.1E+04**			
Lead	7.17E-02 2.30E+01	**3.8E+01**	**7.4E+01**	**1.2E+02**	9.1E+00	**3.5E+01**		**4.7E+01**			
Magnesium	2.56E+00	**7.5E+04**	1.5E+04	1.5E+04	1.6E+04	1.7E+04		**3.0E+04**			

Table 6-9. (continued).

Contaminant	Min EBSL (mg/kg) Background (mg/kg)	ANL-01[b]	ANL-01A[b]	ANL-04[c]	ANL-05[b]	ANL-09[b]	ANL-29	ANL-35[b]	ANL-36	ANL-61A[b]	ANL-62
	1.90E+04										
Manganese	1.44E+01 7.00E+02	**7.7E+02**	**1.2E+03**	2.5E+02	4.2E+02	6.3E+02		**1.2E+03**			
Mercury	6.13E-03 7.40E-02	**3.9E+00**	**8.8E+00**	**3.2E+00**	6.0E-02	**2.7E-01**		**3.1E-01**			
Nickel	2.77E+00 5.50E+01	**9.2E+01**	**9.9E+02**	4.1E+01	3.0E+01	3.6E+01		**6.4E+01**			
Nitrate	3.20E+01 NA	9.5E+00						2.2E+01			
Phosphate	3.34E+01 NA	1.7E+01						2.5E+00			
Potassium	3.34E+01 6.30E+03	**8.1E+03**	4.7E+03	3.2E+03	2.9E+03	5.7E+03		**7.4E+03**			
Selenium	8.34E-02 3.40E-01	**8.4E+00**	**2.1E+00**	**3.5E+00**	—	—		**9.4E-01**			
Silver	1.39E+00 NA	**3.8E+01**	**2.3E+01**	**3.7E+01**	1.2E+00	1.5E+00	**5.4E+03**	**3.5E+02**	**1.7E+01**		
Sodium	1.10E+02 5.20E+02	**1.0E+03**	**1.8E+03**	**1.5E+03**	**2.8E+03**	3.6E+02		**9.4E+02**			
Sulfate	1.77E+01 NA	**3.3E+03**	—		—	—		**1.4E+02**			
Thallium	1.17E-01 6.80E-01	**1.0E+00**	—		—	—		**7.0E-01**			
Vanadium	2.55E-01 7.00E+01	**1.1E+02**	**7.4E+01**	**7.3E+01**	3.1E+01	5.4E+01		**7.2E+01**			
Zinc	6.37E+00 2.20E+02	**5.0E+03**	**8.5E+02**	**2.4E+03**	6.1E+01	1.5E+02		**2.3E+02**			

a. The soil concentrations used for screening are the highest values detected in either the surface or subsurface samples. All values in mg/kg.
b. This site was included in the human health risk assessment, therefore, the concentration used is the 95% UCL.
c. All inorganics observed at ANL-04 will be retained in the WAG 9 ERA as part of the water ingestion pathway. ANL-04 concentrations in a bold typeface are also assessed as part of the food and soil ingestion pathways.
d. NC—Not calculated due to no available data.
e. NA—No background concentration available.

Table 6-10. Retained surface water and sediment contaminants

Industrial Waste Pond and 3 Cooling Tower Blowdown Ditches (ANL-01)		
1,1,1-trichloroethane	Cyanide	Silver
2,4,5-TP	Diethylhexylphthalate	Sodium
Aluminum	Di-n-butylphthalate	Sulfide
Antimony	Fluoride	Sulfate
Arsenic	Iron	Toluene
Barium	Lead	Vanadium
Beryllium	Magnesium	Zinc
Cadmium	Manganese	Cs-137
Calcium	Mercury	Co-60
Chloride	Nickel	Pu-239
Chloroform	Nitrate	Sr-90
Chromium	Phosphate	Th-228
Cobalt	Potassium	Th-230
Copper	Selenium	U-238
ANL Sewage Lagoons (ANL-04)		
Aluminum	Copper	Silver
Antimony	Cyanide	Sodium
Arsenic	Lead	Vanadium
Barium	Magnesium	Zinc
Beryllium	Mercury	Cs-137
Calcium	Nickel	Sr-90
Chromium	Potassium	
Cobalt	Selenium	
Industrial Waste Lift Station Discharge Ditch (ANL-35)		
Copper	Bromoform	

distance to surface springs [over 100 mi (160 km)] (EG&G Idaho, 1993). However, groundwater will be assessed qualitatively when human health has identified it as a concern. Major contaminant classes for all media include metals, organic compounds, and radionuclides.

6.2.7.1 Surface Soil. Contaminated surface soil represents a major source of possible contaminant exposure for WAG 9 ecological components. Surface soil, as defined for use in the INEEL WAG ERAs, includes the uppermost 0.15 m (0.5 ft). Eight of the 9 WAG 9 sites of concern represent sources of surface soil contamination resulting from past contamination.

The ecological pathways/exposure model for WAG 9 contaminated surface soil is shown in Figure 6-4. This model depicts the various mechanisms for surface soil contamination transport. These include:

Wind and water erosion

- Leaching and infiltration
- Plant uptake
- Burrowing animal translocation.

Transportation of contaminated soils through these mechanisms may result in contamination of various other media or secondary sources, including the following onsite and offsite sources:

- Surface water
- Surface soil
- Subsurface soil
- Vegetation.

Receptors having potential for direct exposure to WAG 9 surface soils are presented on Table 6-11. Ecological receptors can be exposed to contaminated media directly through ingestion of vegetation, water, or soil, or through physical contact or inhalation. Inhalation and physical contact, however, are considered to play minor roles in exposure to surface contamination for WAG 9. The functional groups identified as having direct exposure include most terrestrial bird, mammal, reptile, and insect species potentially present in the WAG 9 area.

6.2.7.2 Subsurface Soil. The ecological pathways/exposure model for WAG 9 contaminated subsurface soils is presented in Figure 6-5. Eight of the 9 WAG 9 sites of concern are contaminated subsurface soil sites resulting from buried contaminated soil or sediments, and past surface spills followed by leaching. For the analysis, subsurface soils are defined at depths of 0.5 to 10 ft (15 cm to 3 m). Contaminants in subsurface soil can be transported to ecological receptors by plant uptake and translocation by burrowing animals. Contamination depths greater than 10 ft (3 m) below surface are considered inaccessible to ecological receptors, since this is generally below the root zone of plants and burrowing depth of ground-dwelling animals.

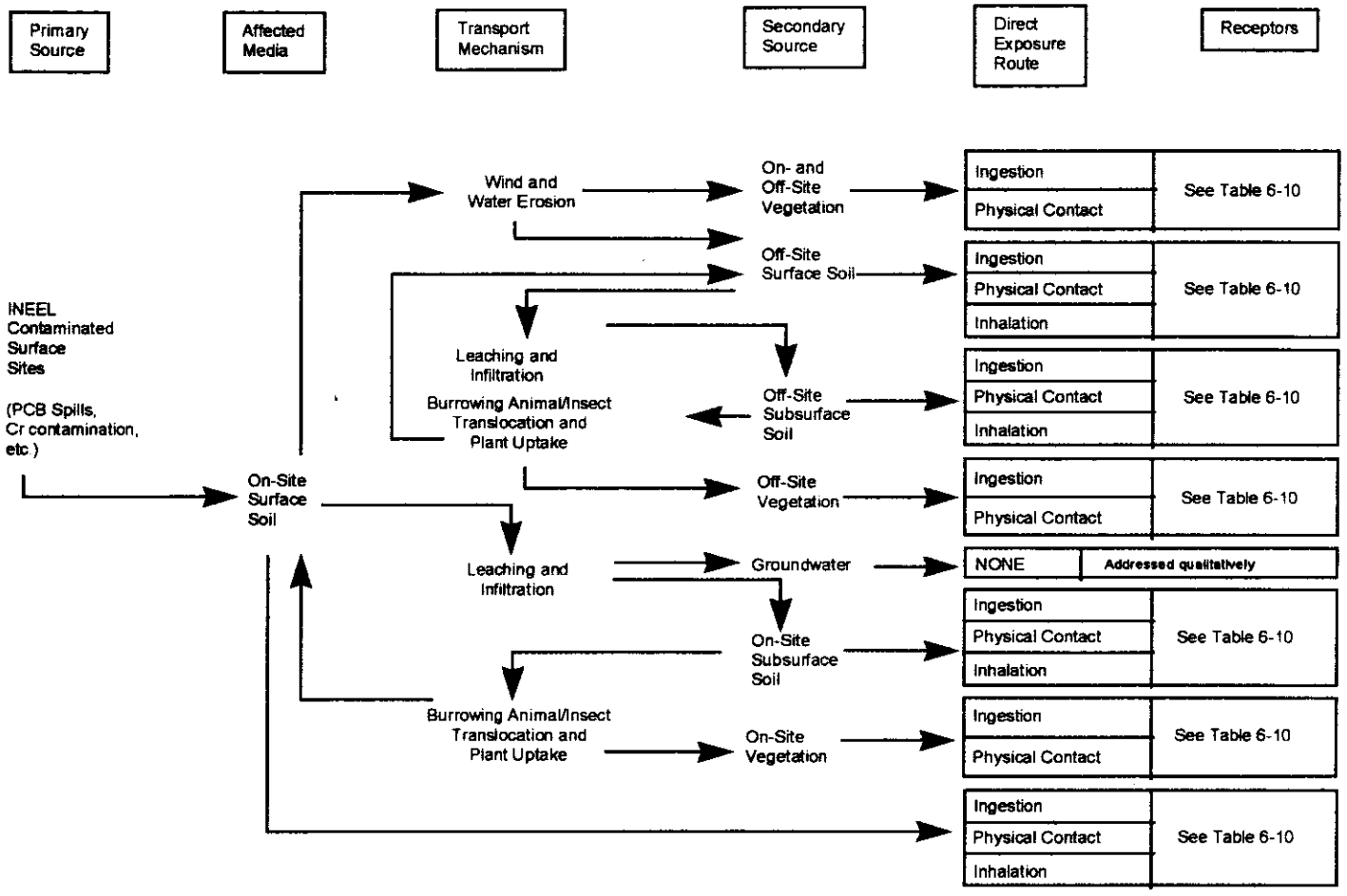

Figure 6-4. Model for ecological pathways and exposure for WAG 9 surface contamination.

Table 6-11. Summary of WAG 9 direct exposure pathways and receptors.

Exposure Medium	Exposure Route	Potential Receptors (functional groups)[a]
Subsurface soil (Direct)	Ingestion (dietary)	AV322A, M122A, M322, M422, R222, R322, terrestrial invertebrates, microorganisms, individual plant species (uptake)
	Physical contact	AV222A, M122A, M322, M422, R222, R322
	Inhalation	Not addressed
Surface soil (Direct)	Ingestion (dietary)	AV122, AV212, AV222, AV322, AV322A, AV422, M122, M122A, M322, M422, M422A, R222, R322, terrestrial invertebrates, microorganisms, individual plant species (uptake)
	Physical Contact	AV122, AV212, AV222, AV322, AV322A, AV422, M122, M122A, M322, M422, M422A, R222, R322, terrestrial invertebrates, microorganisms
	Inhalation	Not addressed
Vegetation (Direct)	Ingestion	AV122, AV143, AV422, M122, M122A, M422, phytophagous insects
	Physical contact	AV122, AV222, AV310, AV322, M122, M122A, terrestrial invertebrates
Surface water (Direct)	Ingestion (dietary)	AV122, AV143, AV212, AV222, AV310, AV322, AV422, M122, M122A, M210A, M322, M422, M422A, R222, R322, aquatic microfauna
	Physical contact	AV143, aquatic microflora/fauna
Sediments (Direct)	Ingestion (dietary)	AV143, benthic invertebrates
	Physical contact	AV143, benthic invertebrates
	Inhalation	Not addressed
Prey (Indirect)	Ingestion	AV212, AV222, AV310, AV322, AV422, M210A, M210, M322, M422, M422A, R222, R322, entomophgous, zoophagous, and saprophagous insects

a. Individual species associated with these groups are listed in Appendix F.

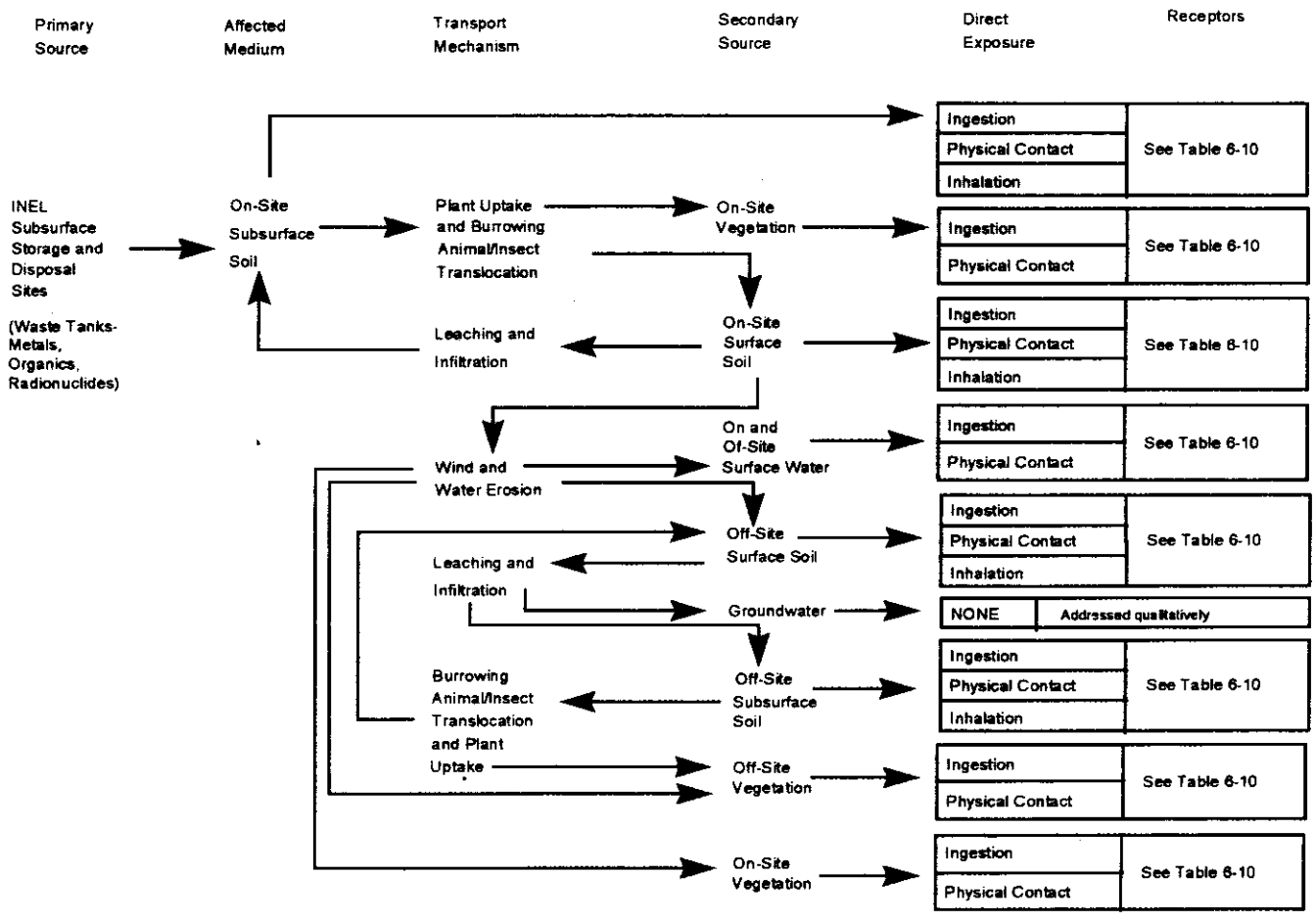

Figure 6-5. Model for ecological pathways and exposure for WAG 9 subsurface storage and disposal sites.

Once contaminated soil is brought close to the surface, transport and exposure scenarios for ecological receptors are the same as for surface soil. For subsurface contamination, inhalation and direct contact (by burrowing animals) are likely more important exposure routes than for surface contamination.

Receptors having potential for direct exposure to WAG 9 subsurface soil contamination are presented on Table 6-10. These receptors include animals dwelling below ground and deep rooting plants. Because subsurface soil contamination may be translocated to the surface by burrowing and plant uptake, other terrestrial species also have some potential for exposure through this pathway.

6.2.7.3 Surface Water. There are three sources of standing water in the WAG 9 assessment site: industrial waste pond and cooling tower blowdown ditches (ANL-01), the sewage lagoons (ANL -04), and the industrial waste lift station discharge ditch (ANL-35). The pathways/exposure model for contaminated surface water sites at the INEEL is shown in Figure 6-6. Ecological receptors having potential for direct exposure to surface water pathways are identified on Table 6-11.

6.2.8 Conceptual Site Model

The pathways/exposure models for surface soil, subsurface soil, and surface water were integrated to produce the WAG 9 conceptual site model (CSM) shown in Figure 6-7. This model reflects both direct (previous sections) and indirect (i.e., predation) receptor exposure pathways for WAG 9 COPCs.

6.2.9 Development of Assessment Endpoints

This section addresses the development of assessment endpoints. Assessment endpoints are "formal expressions of the actual environmental values that are to be protected" (Suter, 1989). Assessment endpoints developed for this WAG ERA are presented on Table 6-12. The endpoints were developed around the protection of INEEL biota represented by functional groups and individual T/E and C2 species known to exist at WAG 9 and identified as having potential for exposure to COPCs. Each T/E species is addressed individually in the risk analysis, whereas potential effects to other receptors of concern are dealt with at the functional group level. Assessment endpoints defined for the WAG 9 ERA reflect INEEL-wide hazard/policy goals discussed in the *INEEL ERA Guidance Manual* (VanHorn et al., 1995) and incorporate the suggested criteria for developing assessment endpoints, including ecological relevance and policy goals (EPA, 1992; Suter, 1993).

These assessment endpoints are the focus of WAG ERA risk characterization and link the measurement endpoints to the WAG ERA goals. The primary objective of this WAG ERA is to identify COPCs and levels of those contaminants that represent potential risk to WAG 9 ecological components. Consequently, toxic effects to ecological components as a result of exposure to COPCs were considered a primary concern for WAG 9 biota. Although adverse effects due to physical stressors are also of concern in evaluating potential risks to INEEL ecological components, these effects are not addressed by the WAG 9 ERA assessment. A health-based approach was used to establish the potential for contaminants to contribute to ecological risk to WAG 9 individuals and populations. The HQ is used to indicate whether or not a potential for adverse effects exists. The use of the HQ as an indicator of effects is discussed in detail in Section 6.4.1.

6.2.10 Measurement Endpoint Selection

This section describes the selection of measurement endpoints for the WAG 9 ERA. Measurement endpoints are measurable responses to ecological receptors to contaminants that can be related to ERA

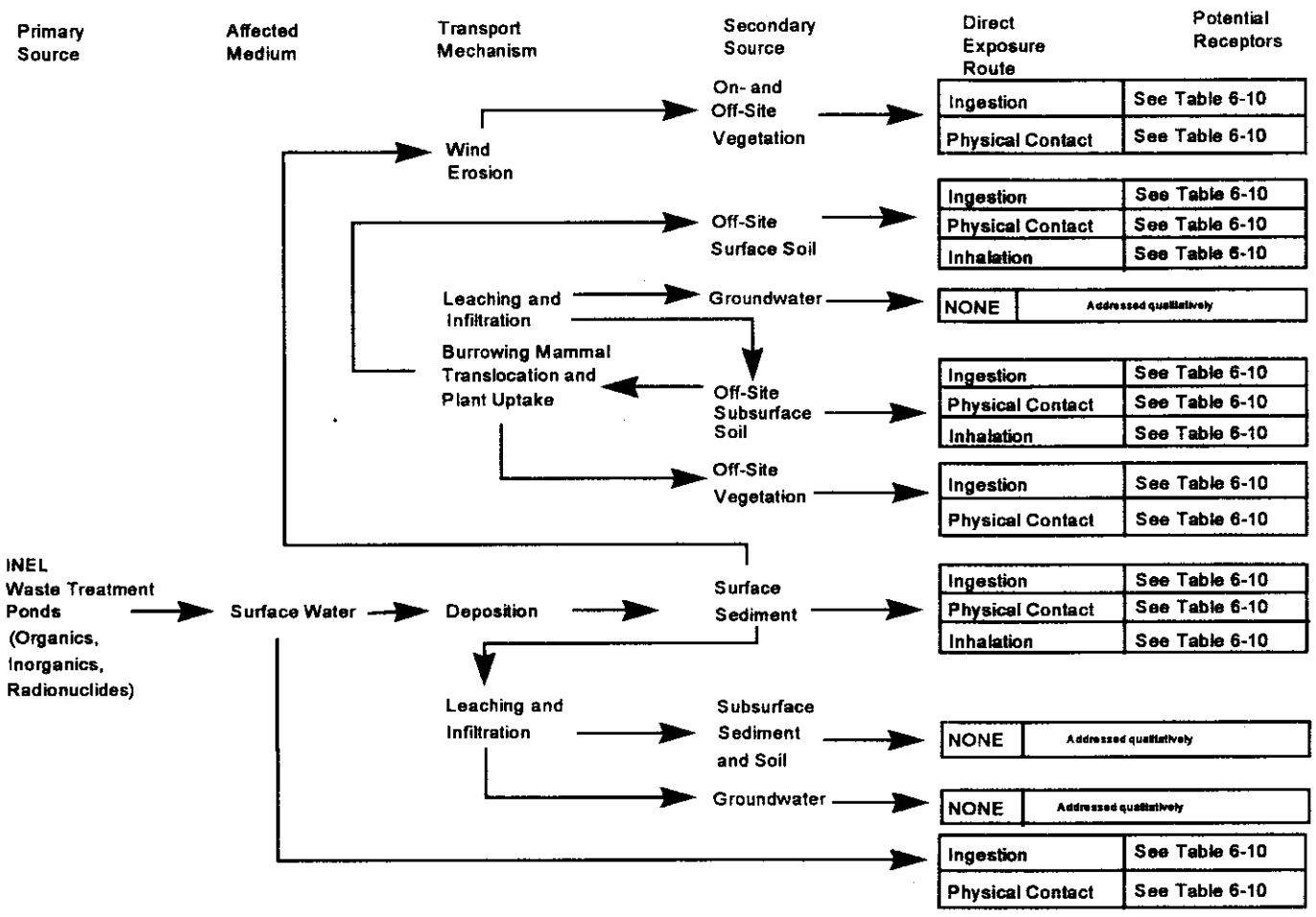

Figure 6-6. Model for ecological pathways and exposure for WAG 9 surface water contamination.

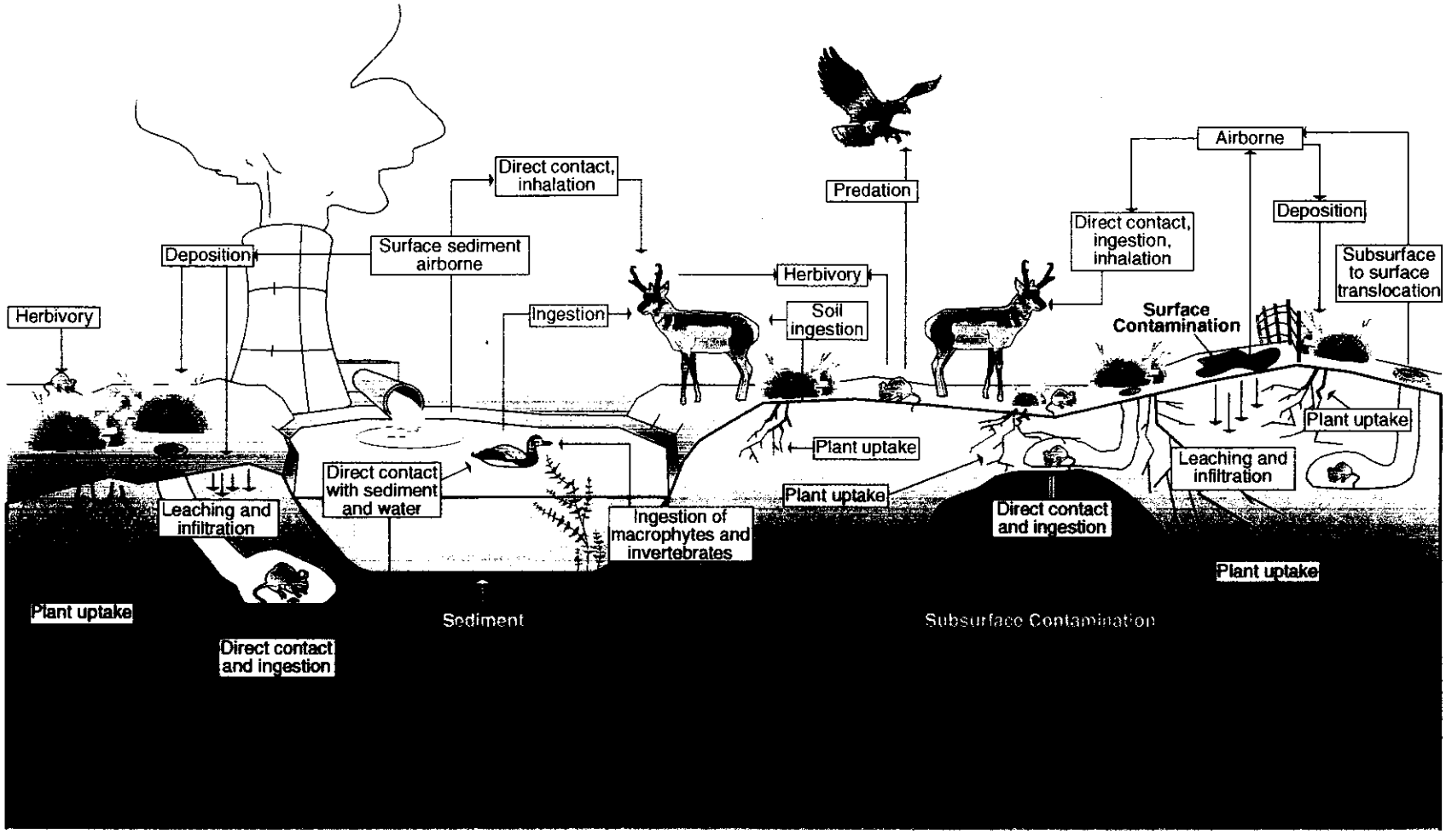

Figure 6-7. WAG 9 ecological conceptual site model.

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Table 6-12. Summary of WAG 9 ERA assessment endpoints.

Management Goals	WAG ERA Endpoint	Indicator of Effects[a]
Maintain INEEL T/E individuals and populations by limiting exposure to organic, inorganic, and radionuclide contamination.	No indication of possible effects to T/E individuals and populations as a result of contaminant exposure: peregrine falcon, northern goshawk, bald eagle, burrowing owl, ferruginous hawk, loggerhead shrike, white-faced ibis, black tern, pygmy rabbit, Townsend's western big-eared bat, long-eared myotis, small-footed myotis, sagebrush lizard, and trumpeter swan individuals and populations (Functional Groups AV310, AV322, AV322A, AV233, AV210, R222, M123 and M210A).	HQ ≥ target value
Maintain INEEL T/E individuals and populations by limiting exposure to physical stressors.	Not addressed by WAG ERA	N/A
Maintain abundance and diversity of INEEL native biota by limiting exposure to organic, inorganic, and radionuclide contamination.	No indication of possible effects to WAG native vegetation communities as a result of contaminant exposure.	HQ ≥ target value
	No indication of possible effects to WAG wildlife populations as a result of contaminant exposure (represented by functional groups identified in the site conceptual model: waterfowl, small mammals, large mammals, song birds, raptors, top predators, invertebrates).	HQ ≥ target value
Maintain abundance and diversity of INEEL native biota by limiting exposure to physical stressors physical stressors.	Not addressed by WAG ERA	N/A

Source: Suter 1993

a. HQ—hazard quotient. The target value is 1 for nonradionuclide contaminants and 0.1 for radionuclide contaminants.

assessment endpoints. For the WAG 9 ERA, the ecological components (flora and fauna) were not surveyed directly. Rather, published references were used as the primary sources of ecological and toxicological data from which measurement endpoints were derived. Values extracted from these references were used to calculate EBSLs for all ecological receptors and to develop TRVs for contaminants.

Table 6-13 summarizes the measurement endpoints developed to address WAG 9 screening-level assessment endpoints. Quantified critical exposure (QCE) levels and adjustment factors (AFs) were constructed from the literature to develop appropriate TRVs for receptors associated with WAG 9 contaminant pathways. Criteria for development of these TRVs are discussed in Section 6.3.4.1. In general, the criteria incorporate the requirements for appropriate measurement endpoints, including relevance to an assessment endpoint, applicability to the route of exposure, use of existing data, and consideration of scale (VanHorn et al. 1995).

Published values for species dietary habits, home ranges, site use, exposure duration, soil ingestion, food digestion, and body weights for the representative species listed on Table 6-14 and the average contaminant concentration in each media were used to calculate doe for each affected receptor.

The measurement endpoints are the modeled dose as compared to the TRVs for each contaminant for each receptor or functional group. The dose was divided by the TRV to produce an HQ for each contaminant and receptor of concern. The HQ is ultimately used to measure whether the assessment endpoint has been attained, that is, no indication of possible effects is determined (HQs are less than target value for all receptors for each contaminant).

6.3 Analysis

The risk analysis step of the WAG 9 ERA involves assessing exposure to contaminants (characterization of exposure) and potential effects of exposure (characterization of effects). These activities are conducted interactively to ensure that the methods used to assess exposure and effects are compatible. Assessing exposure and effects is based on the ecological endpoints and conceptual models derived during the problem formulation presentation.

A primary step in analyzing risk is to determine the potential for site-related contaminants to increase the incidence of adverse effects in exposed populations. The objective of this activity is to estimate the magnitude, frequency, duration, and route of exposure to site-related contaminants by ecological receptors. Accomplishing this task involves completing the following steps:

1. Discuss the factors which influence contaminant fate and transport.

2. Estimate dose for all functional groups and contaminants.

6.3.1 Discuss Contaminant Fate and Transport

This section discusses the behavior and fate of the contaminants in the terrestrial environment. No formal transport and fate modeling was conducted for this WAG ERA. Environmental fate properties are important because they provide information on the environmental behavior of contaminant compounds throughout various environmental media. WAG 9 surface and subsurface soil contaminants identified by the WAG 9 ERA include the following:

Table 6-13. Summary of WAG 9 ERA endpoints.

WAG 9 assessment endpoint	Ecological component	Functional group (other groups represented)	Measurement species (TRV test species)
No indication of possible effects to T/E individuals and populations as a result of contaminant exposure:	Pygmy rabbit	M122A (M123)	Rat, mouse/meadow vole (M122A), deer mouse
	Peregrine falcon, northern goshawk	AV310	Chicken, goshawk, American kestrel/red-tailed hawk (AV322)
	Ferruginous hawk, loggerhead shrike, bald eagle, burrowing owl	AV322, AV322A	Chicken, goshawk, American kestrel/red-tailed hawk (AV322)
	Townsend's western big-eared bat, long-eared myotis, small-footed myotis	M210A (M210)	None located
	Sagebrush lizard	R222	None located
No indication of possible effects to WAG 9 native vegetation communities as a result of contaminant exposure.	Vegetation	Sagebrush, bunchgrass	Bush beans, crop plants
No indication of possible effects to WAG 9 wildlife populations as a result of contaminant exposure (represented by functional groups identified in the site conceptual model: waterfowl, small mammals, large mammals, song birds, raptors, top predators, invertebrates)	Small mammals	M422, M122A (M222, M123)	Rat, mouse/meadow vole (M122A), deer mouse
	Mammalian carnivore/omnivores	M422A, M322	Rat mouse, dog, cat, mink/fox
	Mammalian herbivores	M121 (M122)	Rat, mouse, mule deer/pronghorn
	Avian carnivores	AV322	Goshawk, American kestrel/red-tailed hawk (AV322)
	Avian herbivores	AV122 (AV122)	Chicken, pheasant, quail, passerines/sharp-tailed and ruffed grouse
	Avian insectivore	AV210, AV222 (AV210A, AV221, AV22A)	Chicken, pheasant, quail, passerines/American robin, cliff swallow
	Avian carnivores/omnivores	AV422	Chicken, pheasant, quail passerines
	Mammalian insectivore	M210A (M210)	None located
	Reptiles	R222, R322	None located/Western racer
	Invertebrates	*Phytophagous, saprophagous, entomophagous*	Unidentified

Table 6-14. WAG 9 species parameters.

Functional Groups	PP[a]	PV[b]	PS[c]	SUF	ED[d]	IR (kg/day)[e]	BW (kg)[f]	HR (Ha)[g]	WI (L/day)[h]
Avian herbivores (AV121)	0.00E+00	9.90E-01	1.00E-02	3.67e-04	2.50E-01	3.50E-03	1.29E-02	5.18E+00	3.20E-03
Avian herbivores (AV122)	0.00E+00	9.07E-01	9.30E-02	3.67e-04	1.00E+00	1.46E-03	3.50E-03	5.18E+00	1.33E-03
Avian herbivores (AV132)	0.00E+00	8.20E-01	1.80E-01	1.00e+00	6.50E-01	1.07E-02	7.46E-02	aquatic	1.04E-02
Avian herbivores (AV142)	0.00E+00	9.18E-01	8.20E-02	1.00e+00	2.50E-01	2.75E-02	3.16E-01	aquatic	2.73E-02
Avian herbivores (AV143)	0.00E+00	9.18E-01	8.20E-02	1.00e+00	6.50E-01	2.92E-02	3.47E-01	aquatic	2.90E-02
Avian insectivores (AV210)	9.80E-01	0.00E+00	2.00E-02	2.27e-04	6.50E-01	2.90E-03	1.00E-02	8.38E+00	2.70E-03
Avian insectivores (AV210A)	9.70E-01	0.00E+00	3.00E-02	7.95e-04	6.50E-01	3.89E-03	1.46E-02	2.39E+00	3.48E-03
Avian insectivores (AV221)	9.70E-01	0.00E+00	3.00E-02	1.46e-02	6.50E-01	1.99E-03	6.65E-03	1.30E-01	2.05E-03
Avian insectivores (AV222)	9.07E-01	0.00E+00	9.30E-02	5.00e-03	1.00E+00	3.07E-03	1.09E-02	3.80E-01	2.86E-03
Avian insectivores (AV222A)	9.07E-01	0.00E+00	9.30E-02	4.63e-03	6.50E-01	2.82E-03	1.00E-02	4.10E-01	2.70E-03
Avian insectivores (AV232)	8.20E-01	0.00E+00	1.80E-01	1.00e+00	6.50E-01	1.12E-03	2.32E-03	aquatic	1.01E-03
Avian insectivores (AV233)	8.20E-01	0.00E+00	1.80E-01	1.00e+00	2.50E-01	4.78E-03	2.15E-02	aquatic	4.50E-03
Avian insectivores (AV241)	8.20E-01	0.00E+00	1.80E-01	1.00e+00	2.50E-01	6.41E-03	3.38E-02	aquatic	6.10E-03
Avian insectivores (AV242)	8.20E-01	0.00E+00	1.80E-01	1.00e+00	6.50E-01	1.13E-02	8.10E-02	aquatic	1.10E-02
Avian carnivores (AV310)	9.80E-01	0.00E+00	2.00E-02	8.72e-06	1.00E+00	1.61E-02	1.39E-01	2.18E+02	1.57E-02
Avian carnivores (AV322)	9.80E-01	0.00E+00	2.00E-02	2.11e-04	1.00E+00	7.44E-03	4.25E-02	9.00E+00	7.11E-03
Avian carnivores (AV322A)	9.70E-01	0.00E+00	3.00E-02	1.90e-04	2.50E-01	1.73E-02	1.55E-01	1.00E+01	1.69E-02
Avian carnivores (AV333)	8.20E-01	0.00E+00	1.80E-01	1.00e+00	2.50E-01	1.84E-02	1.71E-01	aquatic	1.81E-02
Avian carnivores (AV342)	9.80E-01	0.00E+00	2.00E-02	1.00e+00	2.50E-01	4.64E-02	7.06E-01	aquatic	4.67E-02
Avian omnivores (AV422)	6.27E-01	2.80E-01	9.30E-02	1.73e-04	1.00E+00	1.13E-02	8.02E-02	1.10E+01	1.09E-02
Avian omnivores (AV432)	5.70E-01	2.50E-01	1.80E-01	1.00e+00	2.50E-01	2.75E-02	3.16E-01	aquatic	2.73E-02
Avian omnivores (AV433)	5.70E-01	2.50E-01	1.80E-01	1.00e+00	2.50E-01	5.33E-02	8.74E-01	aquatic	5.39E-02

Table 6-14. (continued).

Functional Groups	PP[a]	PV[b]	PS[c]	SUF	ED[d]	IR (kg/day)[e]	BW (kg)[f]	HR (Ha)[g]	WI (L/day)[h]
Avian omnivores (AV442)	6.20E-01	2.70E-01	1.10E-01	1.00e+00	1.00E+00	4.41E-02	6.54E-01	aquatic	4.44E-02
Mammalian herbivores (M121)	0.00E+00	9.80E-01	2.00E-02	1.73e-04	2.50E-01	3.14E-01	5.80E+00	1.10E+01	4.82E-01
Mammalian herbivores (M122)	0.00E+00	9.37E-01	6.30E-02	8.26e-03	1.00E+00	3.30E-03	1.10E-02	2.30E-01	1.71E-03
Mammalian herbivores (M122A)	0.00E+00	9.23E-01	7.70E-02	6.33e-03	1.00E+00	4.27E-03	1.57E-02	3.00E-01	2.35E-03
Mammalian herbivores (M123)	0.00E+00	9.23E-01	7.70E-02	9.50e-03	1.00E+00	1.51E-02	8.89E-02	2.00E-01	1.12E-02
Mammalian insectivores (M210)	9.80E-01	0.00E+00	2.00E-02	7.95e-04	5.00E-01	1.43E-03	9.03E-03	2.39E+00	1.43E-03
Mammalian insectivores (M210A)	9.80E-01	0.00E+00	2.00E-02	7.95e-04	2.50E-01	1.43E-03	4.65E-03	2.39E+00	7.88E-04
Mammalian insectivores (M222)	9.76E-01	0.00E+00	2.40E-02	1.53e-02	1.00E+00	1.66E-03	6.00E-03	1.24E-01	9.91E-04
Mammalian carnivore (M322)	9.23E-01	0.00E+00	7.70E-02	1.46e-04	1.00E+00	1.66E-02	1.78E-01	1.30E+01	2.09E-02
Mammalian omnivores (M422)	8.04E-01	1.00E-01	9.40E-02	2.64e-03	1.00E+00	3.06E-03	1.70E-02	7.20E-01	2.53E-03
Mammalian omnivores (M422A)	8.06E-01	1.00E-01	9.40E-02	1.27e-04	1.00E+00	2.60E-01	5.05E+00	1.50E+01	4.25E-01
Reptilian insectivores (R222)	9.76E-01	0.00E+00	2.40E-02	1.62e-02	1.00E+00	5.60E-05	6.61E-03	1.17E-01	0.00E+00
Reptilian carnivores (R322)	9.52E-01	0.00E+00	4.80E-02	6.33e-04	1.00E+00	6.80E-03	1.50E-02	3.00E+00	0.00E+00
Plants	0.00E+00	0.00E+00	1.00E+00		1.00E+00				
Black tern	7.50E-01	0.00E+00	2.50E-01	2.27e-04	2.50E-01	9.84E-03	6.53E-02	8.38E+00	1.36E+02
White-faced ibis	8.90E-01	0.00E+00	1.10E-01	1.00e+00	2.50E-01	4.27E-02	6.22E-01	aquatic	1.00E+00
Northern goshawk	9.80E-01	0.00E+00	2.00E-02	8.92e-06	2.50E-01	6.00E-02	1.05E+00	2.13E+02	5.35E+00
Peregrine falcon	9.80E-01	0.00E+00	2.00E-02	5.74e-05	2.50E-01	4.96E-02	7.82E-01	3.31E+01	3.44E+01
Bald eagle	9.80E-01	0.00E+00	2.00E-02	3.85e-06	2.50E-01	1.60E-01	4.74E+00	4.94E+02	2.31E+00
Ferruginous hawk	9.80E-01	0.00E+00	2.00E-02	3.39e-06	6.50E-01	6.19E-02	1.10E+00	5.60E+02	2.03E+00
Loggerhead shrike	9.80E-01	0.00E+00	2.00E-02	4.16e-04	6.50E-01	7.44E-03	4.25E-02	4.57E+00	2.49E+02
Burrowing Owl	9.70E-01	0.00E+00	3.00E-02	1.90e-04	2.50E-01	1.73E-02	1.55E-01	1.00E+01	1.14E+02

Table 6-14. (continued).

Functional Groups	PP[a]	PV[b]	PS[c]	SUF	ED[d]	IR (kg/day)[e]	BW (kg)[f]	HR (Ha)[g]	WI (L/day)[h]
Pygmy rabbit	0.00E+00	9.80E-01	2.00E-02	6.79e-03	1.00E+00	4.53E-02	4.04E-01	2.80E-01	4.07E+03
Townsend's western big-eared bat	9.90E-01	0.00E+00	1.00E-02	7.95e-04	1.00E+00	2.37E-03	1.10E-02	2.39E+00	4.77E+02
Sagebrush lizard	9.76E-01	0.00E+00	2.40E-02	1.62e-02	1.00E+00	5.60E-05	6.61E-03	1.17E-01	9.74E+03
Trumpeter swan	0.00E+00	9.18E-01	8.20E-02	1.29e-03	2.50E-01	2.75E-01	1.09E+01	1.47E+00	7.75E+02
Small-footed myotis	9.90E-01	0.00E+00	1.00E-02	7.95e-04	1.00E+00	1.44E-03	4.69E-03	2.39E+00	4.77E+02
Long-eared myotis	9.90E-01	0.00E+00	1.00E-02	7.95e-04	1.00E+00	1.77E-03	6.65E-03	2.39E+00	4.77E+02
Amphibians (A232)	9.41E-01	0.00E+00	5.90E-02	1.54e-02	1.00E+00	6.49E-05	8.00E-03	1.24E-01	1.00E+00

PP = percentage of diet represented by prey ingested (unitless). Herbivores = 0% prey, total PV = PV-PS; carnivores = 0% vegetation, total PP - PS): and omnivores =(1.00-PS)/2 for PP and PV.

b. PV = percentage of diet represented by vegetation ingested (unitless).

c. PS = percentage of diet represented by soil ingested (unitless). Soil ingestion from Beyer et al. (1994) and Arthur and Gates (1988) - (pronghorn, jackrabbit).

d. ED = exposure duration (fraction of year spent in the affected area) (unitless). Conventions: Residents - 0.05-1.00 (birds and migratory and transient mammals) 1.00 (small mammals); breeding - 0.05-0.65 (birds and migratory and transient mammals); summer visitors - 0.05-0.25; winter visitors - 0.05-0.25.

e. IR = ingestion rate [derived using allometric equations based on body weight (Nagy, 1987)] (kg/day).

f. BW = receptor-specific body weight (kg). Mammalian body weight primarily from Burt and Grossenheider (1980) and EPA Exposure Factors Handbook (1993) for some species. Avian body weight from Dunning (1993).

g. Home ranges from Hoover and Wills (1987).

h. WI = water ingestion rates derived using allometric equation (Nagy, 1987).

- aluminum
- antimony
- arsenic
- barium
- beryllium
- cadmium
- calcium
- chromium(III)
- chromium(VI)
- cobalt
- copper
- cyanide
- HpCDD
- iron
- lead
- magnesium
- manganese
- mercury
- nickel
- OCDD
- PCBs
- PeCDD
- potassium
- selenium
- silver
- sodium
- sulfate
- thallium
- vanadium
- zinc

Many of the WAG 9 contaminants are metals. Soils represent the most concentrated source of metals in the terrestrial environment. Particulate matter readily sorbs metals, which may complex with various anions such as carbonates and sulfides, modifying their water solubility. Such sorption and complexation (typically) diminishes the bioavailability of metals in soils and sediments or aqueous systems (Adams et al. 1992).

The health risks posed by trace metals in soils are not determined solely by their quantity. A number of contaminant, environmental, and biological conditions and processes influence the accessibility and availability of metals to organisms, and hence their toxicological significance. First, speciation is a major determinant of the fate, bioavailability, absorption, and toxicologic characteristics of metal compounds. Second, the distribution coefficient between soil and water (K_d) depends upon both the properties of the metal and the composition of the soil. This coefficient also governs the bioavailability of a metal to organisms contacting the soil, with weakly bound metals highly bioavailable and more strongly bound metals less bioavailable. Other influential factors include: (1) the characteristics of the interface (e.g., lung, skin, intestine), (2) the reactivity of the metal with the interface, and (3) the concurrent presence of other metals or other substances that may stimulate or inhibit metal uptake.

Factors which influence the fate and transport (and thereby bioavailability) of the WAG COPCs are presented in Sections 6.3.4 and 6.3.5, along with discussions of the ecotoxicological effects and derivation of TRVs for these contaminants.

6.3.2 Determining Exposure

Potential exposures for functional group, T/E, and C2 species were determined based on site-specific life history and feeding habits when possible. Quantification of group and individual exposures incorporated species-specific numerical exposure factors including body weight, ingestion rate, and fraction of diet composed of vegetation or prey, and soil consumed from the affected area. Parameters used to model contaminant intakes by the functional groups are presented in Table 6-13. These values were derived from a combination of parameters that produced the most conservative overall exposure for the group. The functional group parameters in Table 6-13 represent the most conservative combination of percent prey, percent vegetation, percent soil, exposure duration, ingestion rate to body weight ratio, and home ranges from species within the functional group.

Each receptor's diet was assumed to be composed of percentages of two food types (i.e., percentages of either prey or vegetation) to simplify exposure calculations. For example, herbivorous animals are assumed to consume solely contaminated vegetation taken from the WAG 9 area. Vegetation is not broken into seeds vs. vegetative parts to take advantage of the potential differences in plant part uptake. While this is a simplistic and conservative assumption, breaking down the diet of individual species within a functional group in more detail, while warranted, is beyond the scope of a WAG ERA. Most terrestrial receptors incidentally or directly ingest soil and the percent of soil ingested from that affected area was also estimated.

Exposure estimates were corrected for the WAG 9 site areas by the use of site use factors (SUFs). The SUF is the WAG 9 site area (ha) divided by the species' home range (ha) to a maximum of 1.0. The SUF is the proportion of the site area to the home range and is not allowed to be greater than 1.0. Home ranges for the functional groups at WAG 9 are summarized in Table 6-13. However many are unknown and these are defaulted to a SUF of 1.0. A SUF of less than 1 indicates that the home range is larger than the area affected, and it is likely that these functional groups or T/E species consume prey, vegetation, and soil from unaffected areas.

Exposure duration (ED) is based on the migratory pattern of the receptors. This is determined using the status and abundance data compiled for site species (VanHorn et al., 1995). Five status/abundance categories are represented: resident, breeding, summer visitor, migratory, and winter visitor. For year-round residents, ED is assumed to be 1 (i.e., receptors potentially spend up to 100% of the year on the assessment area). For species breeding on-site, the ED is assumed to be 0.65. (i.e., receptors potentially spend up to 65% of the year on the assessment area). For migratory summer and winter visitors, the ED is assumed to be 0.25 (i.e., receptors potentially spend up to 25% of the year on the assessment area). The most conservative ED duration is chosen from the functional group members to represent the functional group ED.

Food intake rates (g dry weight/day) for passerine birds, nonpasserine birds, rodents, herbivores, all other mammals, and insectivorous reptiles can be estimated using the following allometric equations (Nagy 1987). The equation for insectivorous reptiles can be conservatively assumed to be applicable to the carnivorous reptiles (R322). Because allometric equations may apply to different species within a group, the equations representative of all mammals and aviars were used to calculate the IR for the functional groups. Exposure of each functional group was calculated using best available estimates for species-

specific exposure parameters. Each of the receptors was evaluated individually. Potential exposures for these species was determined based on the species' life history and feeding habits. Quantification of exposures used species-specific numerical exposure factors including body weight, ingestion rate, and fraction of diet composed of vegetation or prey, and soil consumed from the affected area. Species parameters used to model intakes by the functional groups are presented in Table 6-13. These values are derived from the various key species in the functional groups. The parameters in Table 6-13 are the maximum percent prey, percent vegetation, percent soil, exposure duration, and the minimum ingestion rate to body weight ratio and home ranges for each functional group because these values were the most conservative. Percent soil ingestion rate values come from the *Wildlife Exposure Factors Handbook* (EPA 1993a) and Beyer et al. (1994) and site specific data where available.

$$Food\ intake\ rate = 0.398\ BW^{0.850}\ (passerines) \quad (6\text{-}1)$$

$$Food\ intake\ rate = 1.110\ BW^{0.445}\ (desert\ bird) \quad (6\text{-}2)$$

$$Food\ intake\ rate = 0.648\ BW^{0.651}\ (nonpasserines) \quad (6\text{-}3)$$

$$Food\ intake\ rate = 0.583\ BW^{0.585}\ (rodents) \quad (6\text{-}4)$$

$$Food\ intake\ rate = 0.577\ BW^{0.727}\ (herbivores) \quad (6\text{-}5)$$

$$Food\ intake\ rate = 0.15\ BW^{0.874}\ (desert\ mammals) \quad (6\text{-}6)$$

$$Food\ intake\ rate = 0.013\ BW^{0.773}\ (insectivorous\ reptiles) \quad (6\text{-}7)$$

where BW = body weight in grams.

6.3.2.1 Exposure to Nonradiological Contaminants. The exposure equation used to calculate average daily intake is used to calculate the dose to functional group and T/E species. For example, dose (intake) in mg/kg body weight-day can be estimated using the following equation, as adapted from EPA's Wildlife Exposure Factors Handbook (EPA 1993):

$$EE_{tot} = \frac{[(PP \times CP) + (PV \times CV) + (PS \times CS)] \times IR \times ED \times SUF}{BW} \quad (6\text{-}8)$$

where

EE_{tot} = estimated exposure from all complete exposure pathways (mg/kg body weight-day)

PP = percentage of diet represented by prey ingested (unitless)

CP = concentration of contaminant in prey item ingested (mg/kg)

PV = percentage of diet represented by vegetation ingested (unitless)

CV = concentration of contaminant in vegetation ingested (mg/kg)

PS = percentage of diet represented by soil ingested (unitless)

CS = concentration of contaminant in soil ingested (mg/kg)

IR = ingestion rate (kg/day), food intake rate (g/day) divided by 1,000 g/kg

ED = exposure duration (fraction of year spent in the affected area) (unitless)

BW = receptor-specific body weight (kg)

SUF = site usage factor (site area divided by home range; cannot exceed 1) (unitless).

The concentration of contaminant in prey can be estimated using the equation:

$$CP = CS \times BAF \tag{6-9}$$

where

CP = concentration in prey item ingested (mg/kg)

CS = concentration of contaminant in soil (mg/kg)

BAF = contaminant-specific bioaccumulation factor (unitless).

$$CV = CS \times PUF \tag{6-10}$$

The concentration of contaminant in vegetation (CV) can be estimated using the equation:

where

CV = concentration in vegetation (mg/kg)

CS = concentration of contaminant in soil (mg/kg)

PUF = contaminant-specific plant uptake factor (unitless).

Finally, burrowing and nonburrowing animals are potentially exposed to different soil concentrations. In order to account for this, nonburrowing animals are expected to only ingest surface soils; however, their prey are still considered to be potentially exposed to subsurface conditions.

Combining Equations 6-8 through 6-10 gives the following total dose to nonradiological contaminants in mg/kg body weight-day:

$$EE_{tot} = [(PP \times BAF + PV \times PUF + PS) \times CS_g \times IR + WI \times CW]\left(\frac{ED \times SUF}{BW}\right) \tag{6-11}$$

for burrowers, and

$$EE_{tot} = \{[(PP \times BAF + PV \times PUF) \times CS_g + CS_s \times PS] \times IR + WI \times CW\} \times \left(\frac{ED \times SUF}{BW}\right) \quad (6\text{-}12)$$

for nonburrowers, where

WI = water ingestion rate (L/d)

CS_s = surface soil concentration (mg/kg)

CS_g = the greater of the surface and subsurface soil concentrations (mg/kg)

CW = concentration of contaminant in water (mg/L).

The water ingestion is found from the following equations:

$$WI = 0.059\ BW^{0.67}\ \textit{(for birds)} \quad (6\text{-}13)$$

$$WI = 0.099\ BW^{0.90}\ \textit{(for mammals)} \quad (6\text{-}14)$$

Due to the complexity of water ingestion by reptiles, no general reptilian water ingestion equation is available. It is assumed here that desert reptiles, such as those found at the INEEL, get their water solely from prey.

Water concentrations of contaminants at ANL-01, ANL-04, and ANL-35 came from a variety of documents and databases. There is no surface water data for some of the COPCs. In these cases, sediment concentrations were used to derive water concentrations. That is, when a contaminant was observed in the sediments, but not in the effluent data, the sediment concentrations were used to derive a water concentration. When water is present, the ditches may support aquatic species such as frogs and aquatic invertebrates. These populations cannot be considered indicative of a viable ecosystem, as they are periodically exterminated when the ditches dry up. Therefore, these ponds were not evaluated as aquatic habitat, and were assessed from a wildlife drinking water perspective only.

Some of the potential contaminants identified in the effluent monitoring samples are not considered in this ecological risk assessment. These include the essential elements Ca and K. These elements do not have toxicological benchmarks for wildlife. These elements are essential for animal growth and survival, and are therefore readily metabolized and generally non-toxic. When a contaminant is present in the sediments but is not present in the surface water results, a surface water concentration was calculated by assuming that the partitioning of the contaminant in the sediment and water reaches equilibrium. In this case, the equation used to calculate nonradionuclide concentrations in surface water from concentrations in sediment is:

$$CW = \frac{1{,}000ml/l \times Cs \times \rho \times 1\ kg/1{,}000\ g}{K_d \rho + \theta} \quad (6\text{-}15)$$

where

Cs = concentration of the nonradionuclide in sediment (mg/kg)

ρ = soil bulk density (g/cm^3) - assumed to be 1.5 g/cm^3

K_d = partition coefficient between soil and water ([mg/g soil]/[mg/cm^3 water])

θ = volumetric water concentration (m^3/m^3) - assumed to be 0.3 m^3/m^3.

When no K_d value is available, the recommended Track 2 values were used (DOE, 1993). When even these were not available, a conservative value of zero was used.

The following functional groups and T/E species are considered burrowers: AV212, M122A, M210A, M222, M322, M422, M422A, R222, R322, burrowing owl, pygmy rabbit, Townsend's western big-eared bat, and the sagebrush lizard.

Contaminant-specific PUFs and BAFs for nonradionuclides contaminants are presented in Table 6-15. PUFs for all metals are taken from Baes et al. (1984). The PUF and BAFs for organics are estimated using the Travis and Arms (1988) equation of 1.588-0.578 log K_{ow} and -7.735 + 1.033 log k_{ow}, respectively. Log partitioning coefficients (K_{ow}s) were taken from Montgomery and Welkom (1990).

6.3.2.2 Dose for Radiological Contaminants. *The following sections describe the estimation of dose for radiological contaminants. Both internal and external radiation dose development are described separately.*

Internal Radiation Dose—Internal radiation dose estimates are calculated by assuming that the steady-state whole body concentration is equivalent to the steady-state concentration of radionuclides in reproductive organs using the equation:

$$Dose = \frac{TC \times ED \times SUF \times AE \times 3{,}200\ dis/day - pCi}{6.24 \times 10^9\ MeV/g\ tissue - Gy} \qquad (6\text{-}16)$$

where

$Dose$ = radiation dose estimate (Gy/day)

TC = tissue radionuclide concentration (pCi/g)

ED = exposure duration (fraction of year spent in affected area) (unitless)

AE = average absorbed energy (MeV/dis).

Table 6-15. PUFs and CFs for WAG 9 nonradionuclide contaminants (unitless).

	PUF[a]	BAF[b] for Insectivores	BAF[c] for Carnivores	BAF[d] for Omnivores
Inorganics[e]				
Aluminum	4.0E-03	1.0E+00	4.0E-03	1.0E+00
Antimony	2.0E-02	1.0E+00	6.0E-03	9.0E-01
Arsenic	4.0E-02	1.0E+00	4.0E-02	1.0E+00
Barium	1.5E-01	1.0E+00	1.5E-02	1.0E+00
Beryllium	1.0E+00	1.0E+00	1.0E-02	1.0E+00
Cadmium	5.5E-01	1.1E+00	1.9E+00	1.9E+00
Chloride	1.0E+00	1.0E+00	1.0E+00	1.0E+00
Chromium	7.5E-03	6.0E-02	2.0E-01	2.0E-01
Cobalt	1.0E+00	1.0E+00	2.0E-02	1.0E+00
Copper	4.0E-01	1.0E+00	2.0E-01	1.0E+00
Fluoride	1.0E+00	1.0E+00	1.0E+00	1.0E+00
Cyanide	1.0E+00	1.0E+00	1.0E+00	1.0E+00
Lead	4.5E-02	3.0E-01	6.0E-01	6.0E-01
Magnesium	1.0E+00	1.0E+00	1.0E+00	1.0E+00
Manganese	1.0E+00	1.0E+00	2.5E-01	1.0E+00
Mercury	9.0E-01	4.0E-01	7.0E-01	7.0E-01
Nickel	6.0E-02	1.0E+00	6.0E-03	1.0E+00
Selenium	2.5E-02	1.0E+00	2.5E-02	1.0E+00
Silver	4.0E-01	1.0E+00	4.0E-01	1.0E+00
Sodium	1.0E+00	1.0E+00	1.0E+00	1.0E+00
Sulfate	1.0E+00	1.0E+00	1.0E+00	1.0E+00
Thallium	4.0E-03	1.0E+00	4.0E-03	1.0E+00
Vanadium	5.5E-03	1.0E+00	5.5E-03	1.0E+00
Zinc	1.5E+00	1.0E+00	7.0E-01	1.0E+00
Organics[f]				
PCBs	2.9E-03	1.3E-03	1.3E-03	1.3E-03
TCDD[g]	1.0E+00	1.0E+00	1.0E+00	1.0E+00

a. PUF = Plant uptake factor, appropriate for use with AV100 and M100 Functional Groups.

b. BAFs for insectivores, appropriate for A v200 and M200 Functional Groups.

c. BAFs for carnivorous, appropriate for AV300 and M300 Functional Groups.

d. BAFs for omnivores, appropriate for AV400 and M400 Functional Groups.

e. Values and or literature (Appendix H) for inorganics come from Baes et al., (1984).

f. Values for organics come from allometric equations presented in Travis and Arms (1988).

g. PUF and BAFs calculated for TCDD, were used for HpCDD, OCDD, and PeCDD.

Since tissue levels of radionuclides are derived by multiplying the concentration of radionuclide in soil by a radionuclide-specific concentration factor (CF) for all terrestrial animals or terrestrial plants, the above equation can be rewritten as:

$$Dose = \frac{CS \times CF \times ED \times SUF \times AE \times 3{,}200\ dis./day\text{-}pCi}{6.24 \times 10^{9}\ MeV/g\ tissue\text{-}Gy} \qquad (6\text{-}17)$$

where

Dose = internal radiation dose estimate (Gy/day)

CS = concentration of contaminant in soil ingested (pCi/g soil)

CF = concentration factor [unitless; determined by (pCi/g tissue)/(pCi/g soil)]

ED = exposure duration (unitless)

AE = average absorbed energy (MeV/dis).

For alpha-emitting radionuclides, the dose is multiplied by 20 to account for the increased quality of alpha radiation.

As mentioned in Section earlier, water ingestion of radionuclides may occur. To get the water ingestion of contaminants, a simple differential equation was used.

$$\frac{dTC}{dt} = I - \lambda_1(TC) - \lambda_2(TC) - L \qquad (6\text{-}18)$$

where

TC = tissue concentration (pCi/g tissue)

I = intake [(pCi/L) (L/g tissue-day)]

λ_1 = radiological decay constant (1/day)

λ_2 = biological loss constant (1/day)

L = other loss (e.g., through urination) [(pCi/L) (L/g tissue-day)].

Conservatively assuming L = 0 and solving for TC at equilibrium (i.e., dTC/dt = 0) gives

$$TC = \frac{I}{\lambda_1 + \lambda_2} \qquad (6\text{-}19)$$

The daily ingestion rate of the radionuclide from water, I, is calculated as

$$I = \frac{CW \times WI}{BW \times 1{,}000\ g/kg} \tag{6-20}$$

where

CW = concentration of the radionuclide in water (pCi/L)

WI = water ingestion rate (L/d)

BW = body weight (kg)

So the tissue concentration due to water ingestion is:

$$TC = \frac{CW \times WI}{BW \times (\lambda_1 + \lambda_2) \times 1{,}000\ g/kg} \tag{6-21}$$

Because water concentrations are not available for some of the radionuclides in ANL-01 and for the radionuclides in ANL-04, these concentrations had to be derived from the available sediment concentrations. The equation used to calculate water concentrations (pCi/L) from the sediment concentration is:

$$CW = \frac{1{,}000\ ml/L \times Cs \times \rho}{K_d \rho + \theta} \tag{6-22}$$

where

Cs = concentration of the radionuclide in sediment (pCi/g)

ρ = soil bulk density (g/cm^3)—assumed to be 1.5 g/cm^3

K_d = partition coefficient between soil and water [(pCi/g soil) / (pCi/cm^3 water)]

θ = volumetric moisture content (m^3/m^3)—assumed to be 0.3 m^3/m^3

The K_d, λ_1, λ_2, ANL-04 sediment concentrations (Cs), and calculated water concentrations (CW) are shown in Table 6-16. The biological loss constants, λ_2, are based on data from Schultz and Whicker (1982), as discussed below. The radiological decay constants are derived directly from the radionuclide half-life data as

$$\lambda_1 = \frac{\ln 2}{t_{1/2}} \tag{6-23}$$

where $t_{1/2}$ is the half-life (d) from Friedlander et al. (1981).

The biological loss constants are calculated from the observed retention times of radionuclides as documented in Schultz and Whicker (1982). The retention times represent the amount of time the

Table 6-16. Parameters used for calculation of water concentrations.[a]

Radionuclide	K_d	l_1	Biological half-life	l_2	CS	CW
Cs-137	1.1E+3	6.31E-05	1 yr	1.90E-03	3.20E-01	2.91E-01
Sr-90	2.4E+1	6.66E-05	5 yr	3.80E-04	4.21E+0	1.74E+2

a. Symbol definitions and units are given in the text.

radionuclide will remain in the body tissues. Schultz and Whicker (1982) gives qualitative estimates of these times. If the retention time is high (on the order of years) a "biological half-life" of 5 years was assigned to the radionuclide. If the retention time was moderate (months), a biological half-life of 1 year was assigned to the radionuclide. If the retention time was low (days), a biological half-life of 30 days was assigned to the radionuclide. The biological loss constants, λ_2, are then calculated as

$$\lambda_2 = \frac{\ln 2}{t_{biol/2}} \qquad (6\text{-}24)$$

where $t_{biol/2}$ is the biological half-life (d).

As with the nonradiological dose equation, burrowing and nonburrowing animals are handled slightly differently to account for exposure to different soil concentrations. The total internal radiation dose rate (Gy/day) for burrowing animals is given by:

$$Dose_{internal} = \left(CS_g \, x \, CF + \frac{CW \, x \, WI}{BW \, x \, (\lambda_1 + lambdasub2) \, x \, 1{,}000} \right) x \, SUF \, x \, ED \, x \, AE \, x \, \frac{3{,}200}{6.24 x 10^9} \qquad (6\text{-}25)$$

and for nonburrowers is:

The average absorbed energy, AE, is calculated by first determining the average decay energy per disintegration, $\bar{E}$, with units of MeV/dis. Only radiations with an intensity of 1% or greater are considered, and Auger and conversion electrons are not considered. The $\bar{E}$ are calculated using the following equation (Kocher 1981):

$$\overline{E} = \sum_i Y_i E_i \qquad (6\text{-}26)$$

where

Y_i = yield or intensity

E_i = energy of radiation, for ß = average energy (MeV/dis).

The average decay energy per disintegration is calculated separately for α/β and γ decay, producing respectively $\bar{E}_{\alpha/\beta}$ and $\bar{E}_\gamma$. The average absorbed energy, *AE*, is then the sum of the two products calculated by multiplying the average decay energies by a factor accounting for the fraction absorbed and a factor accounting for the quality of the radiation.

$$AE = (FA_{\alpha/\beta} \times QR_{\alpha/\beta} \times \overline{E}_{\alpha/\beta}) + (FA_{\gamma} \times QR_{\gamma} \times \overline{E}_{\gamma}) \quad (6\text{-}27)$$

where

FA = fraction of energy absorbed (unitless)

QR = quality of radiation (unitless).

BAFs for radionuclides are presented in Table 6-17. Animal BAFs were obtained from the literature and site-specific data. The BAF for Sr-90 (used for mammalian herbivores) was calculated by dividing the deer mouse carcass concentration (Arthur et al., 1987) by the soil concentration (Arthur and Markham, 1983). A second value for the BAF for Sr-90 for mammalian herbivores was derived by dividing the pronghorn carcass concentration by the soil concentration (Markham et al., 1980). The most conservative of these two values was used. If BAF values were not available in the literature then the CF was assumed to be 1.

External Radiation Dose—External dose is derived using formulas outlined in Shleien (1992). Dose rate to tissue in an infinite medium uniformly contaminated by a gamma emitter is calculated as:

$$\dot{D} = \frac{2.12 \times \overline{E} \times C \times ED \times SUF}{\rho} \quad (6\text{-}28)$$

where

$\dot{D}$ = external dose rate to tissue (rads/hr)

$\overline{E}$ = average gamma decay energy per disintegration (MeV/dis)

C = concentration of contaminant (μCi/cm^3)

ρ = density of the medium (g/cm^3).

The density, ρ, of soil is assumed to be 1.68 g/cm^3. Since soil concentrations are given in pCi/g, they need to be converted to μCi/g by dividing by 10^6. Finally, the dose in Equation (28-27) is converted to Gy/day using the conversion factor 0.24. The resulting equation for burrowing animals is:

$$Dose_{external} = (0.24 \times 2.12 \times \overline{E} \times CS_g \times ED \times SUF) / (1.68 \times 10^6) \quad (6\text{-}29)$$

and for nonburrowing animals is:

$$Dose_{external} = (0.24 \times 2.12 \times \overline{E} \times CS_s \times ED \times SUF) / (2 \times 1.68 \times 10^6) \quad (6\text{-}30)$$

This equation conservatively estimates the dose to burrowing terrestrial functional groups. This equation also conservatively reflects that these functional groups spend 100% of their time with external exposure. For the nonburrowing functional groups, it is conservatively assumed that they are exposed to 50% (hemisphere) of radiation.

Table 6-17. WAG 9 radiological BAFs (unitless).

Parameter	H-3	Co-60	Cs-137	Pu-239	Th-228	Th-230	Sr-90	U-238
Avian herbivores (AV122)	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Avian herbivores (AV143)	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Avian insectivores (AV212)	1.00E+00	5.00E-01	5.00E-01	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Avian insectivores (AV222)	1.00E+00	5.00E-01	5.00E-01	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Avian carnivores (AV310)	1.00E+00	1.00E+00	2.00E+00	1.00E-01	1.00E+00	1.00E+00	1.10E+00	1.00E-01
Northern goshawk	1.00E+00	1.00E+00	2.00E+00	1.00E-01	1.00E+00	1.00E+00	1.10E+00	1.00E-01
Avian carnivores (AV322)	1.00E+00	1.00E+00	2.00E+00	1.00E-01	1.00E+00	1.00E+00	1.10E+00	1.00E-01
Ferruginous hawk	1.00E+00	1.00E+00	2.00E+00	1.00E-01	1.00E+00	1.00E+00	1.10E+00	1.00E-01
Avian omnivores (AV422)	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Avian omnivores (AV442)	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Mammalian herbivores M122)	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Mammalian herbivores M122A)	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Pygmy rabbit	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Mammalian insectivores M210)	1.00E+00	5.00E-01	5.00E-01	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Mammalian insectivores M210A)	1.00E+00	5.00E-01	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Townsend's western big-eared bat & small-footed myotis	1.00E+00	1.00E+00	2.00E+00	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Mammalian insectivores (M222)	1.00E+00	1.00E+00	5.00E-01	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Mammalian carnivores (M322)	1.00E+00	1.00E+00	2.00E+00	1.00E-01	1.00E+00	1.00E+00	1.54E+00	1.00E-01
Mammalian omnivores (M422)	1.00E+00	1.00E+00	5.00E-01	1.00E+00	1.00E+00	1.00E+00	1.07E+00	1.00E+00
Mammalian omnivores (M422A)	1.00E+00	1.00E+00	5.00E-01	1.00E+00	1.00E+00	1.00E+00	1.07E+00	1.00E+00
Reptilian insectivores (R222)	1.00E+00	5.00E-01	5.00E0-1	1.00E+00	1.00E+00	1.00E+00	1.54E+00	1.00E+00
Reptilian carnivores (R322)	1.00E+00	1.00E+00	2.00E+00	1.00E-01	1.00E+00	1.00E+00	1.54E+00	1.00E-01

6.3.2.3 Uncertainty Associated with Functional Groups. The selection of receptor parameters used is designed to ensure that each of the members of the functional groups is conservatively represented. Since all members of a functional group are considered similar, it is reasonable to assume that all members of a group will be equally exposed to site-related contaminants. Quantification of dose for each functional group is expected to provide sufficient data to assess the general condition of the ecosystem and to be adequately protective of the majority of species potentially inhabiting WAG 9. In addition, sensitive species are included on the list of receptors for which dose is calculated. Hence, uncertainty associated with the selection of receptor parameters is expected to minimally influence dose estimates.

6.3.2.4 Uncertainty Associated with the Ingestion Rate Estimation. Using food intake rates in dry weight/day may overestimate intake rates since dry weights will contain more contamination/unit vegetation. Intake (ingestion) estimates used for the terrestrial receptors are based upon data in the scientific literature, when available. Food ingestion rates are calculated by use of allometric equations reported in the Nagy (1987). Uncertainties associated with the use of allometric equations could result in either an over-estimation or underestimation of the true dose rate, because not all of these values are known exactly.

6.3.2.5 Uncertainty Associated with the Receptor Site Usage. The calculation of dose incorporated the probability that the receptors may use or inhabit each site. The SUF is defined as the affected area (ha) divided by the home range (ha) of the receptor. If a given receptor's home range is larger than the affected area, then it is reasonable to assume that the receptor may not spend 100% of its life within the site area. Incorporation of the site use factor adjusts the dose to account for the estimated time the receptor spends on the site. The less time spent on the site the lower the dose. Home ranges for several functional groups are unknown and in these cases the SUF equals 1. This may overestimate the potential exposure to these receptors.

6.3.2.6 Uncertainty Associated with the PUFs and BAFs. Using PUFs to estimate plant concentrations has the advantages that it is easy to use and requires minimum data inputs (i.e., the measured or estimated concentration of metal in soil and a PUF taken from the literature). A PUF of 0.01 indicates that the plant concentration should be 1/100th of the total concentration in soil. For this WAG ERA, PUFs for metals are taken from Baes et al., (1984). Although preference is given to studies that reported the steady-state concentration of metals in plants at edible maturity, various soil properties are not considered and data for numerous plant species (both animal feeds and those consumed by humans) are combined. However, root uptake of metals is a complex process that depends on various soil properties (e.g., pH, CEC, and organic matter content) as well as the metal and type of plant involved. Therefore, the use of generic or crop-specific PUFs taken from the literature may not accurately estimate the concentration of metals in plants for all environmental conditions and species that may occur on WAG 9. The PUF for organics is estimated using the geometric mean regression equation developed by Travis and Arms (1988) and using log K_{ow} values. The reliability of estimated PUFs is directly related to the reliability of the K_{ow} values used for the organics. Since K_{ow} values can vary greatly, use of the Travis and Arms (1988) equation to estimate a PUF for organics may over- or underestimate the true dose for organics.

There is a great deal of uncertainty associated with the BAFs used to calculate dose. Very few BAFs are available in the scientific literature, since they must be both contaminant- and receptor-specific. BAFs used for metals are discussed in Appendix H. The regression equation (Travis and Arms 1988) was used to calculate BAFs for the organic contaminants at WAG 9. It is assumed that terrestrial receptors of concern accumulate metals and organics in a similar way and to a comparable degree as beef and dairy cattle. In the absence of specific BAFs, a value of 1 was assumed. This assumption could over- or underestimate the true dose from the contaminant, and the magnitude of error cannot be quantified. The terrestrial receptors of concern for WAG 9 may accumulate organics to a much larger or smaller degree than beef and dairy cattle, and using the regression equation (Travis and Arms 1988) could therefore overestimate or underestimate the true dose from the COPCs. Also the use of BAFs as discussed in Appendix H could result in over and/or underestimating dose to ecological receptors at the site in the absence of site-specific data.

6.3.2.7 Uncertainty Associated with Soil Ingestion. The exposure assessment incorporates percentage of soil ingested by each representative of the functional groups. Although food ingestion rates have the greatest effect on intake estimates, soil ingestion rates could also influence intake rates and, therefore, dose estimates. The EPA Wildlife Exposure Factors Handbook (EPA, 1993) was used to assign soil ingestion parameters to four of the 12 functional groups and the percent soil ingested was assigned to one species (Arthur and Gates 1988). Where information did not exist in the literature on soil ingestion rates for terrestrial biota, soil ingestion rates are assumed to be 2% of the food ingestion rate for all burrowing mammals and birds who consume whole terrestrial prey and 1% for all other receptors. Estimating the percent soil ingested may over- or underestimate the dose since the effect of the estimated

values on the overall dose outcome is dependent on the concentration of contaminant in the media of concern.

6.3.2.8 Uncertainty Associated with the Radiological Equation. Equation (6-16) was taken from an International Atomic Energy Agency (IAEA) report titled Effects of Ionizing Radiation on Plants and Animals at Levels Implied by Current Radiation Protection Standards (IAEA 1992). This equation was for the estimation of dose rates to plants. For the estimation of dose rates to animals, the IAEA recommends multiplying the internal Cs-137 dose rates to animals by a factor of 10 to account for bioconcentration mechanisms (e.g., carnivory, or consumption of certain plants) for species not incorporated into the human food-chain calculation. This may under- or overestimate the dose from those particular radionuclides.

6.3.3 Ecological Effects Assessment

Ecological effects assessment consists of three elements:

- Selecting QCE levels
- Developing AFs
- Developing TRVs.

The following sections contain a general description of the procedures of ecological affects assessment and discussions of each of the three elements.

6.3.3.1 General Procedures of the Ecological Effects Assessment. A TRV is defined as a dose for a receptor (including sensitive subgroups such as taxa under regulatory protection) that is likely to be without appreciable risk of deleterious effects from chronic exposure. Application of toxicity data derived from surrogate species introduces uncertainty into the risk assessment. The magnitude of this uncertainty depends largely upon: (1) the degree of taxonomic difference between the key and test species; (2) the conditions under which the toxicity data are obtained; and (3) the endpoint of interest [e.g., chronic lowest-observed-adverse-effect-level (LOAEL) or no-observed-adverse-effect-level (NOAEL)] and the endpoint measured (e.g., death).). AFs are applied in the development of the TRVs in an attempt to offset the uncertainties associated with extrapolation of toxicity information from literature to site conditions.

The approach for TRV derivation used in this WAG ERA was developed by Ludwig et al., (1994) for use at the Rocky Mountain Arsenal Superfund site in Commerce City, CO, and is generally based on the EPA reference dose approach as modified by Lewis et al., (1990). It is predicated on the development and application of AFs, which are intended to explicitly account for variations and uncertainties in the data and necessary extrapolations from it. The types of variation and extrapolation uncertainties explicitly quantified are:

- Variation in sensitivity among the members of a receptor population
- Uncertainty in extrapolating data from one taxon to another
- Uncertainty in using various effect levels to estimate no-effect levels receptors

- The inability of any single study to adequately address all possible adverse outcomes in a wild receptor population.

The approach of Ludwig et al. (1994) offers several distinct advantages. By carefully identifying the specific types of adjustments needed in the extrapolation, this method permits maximum resolution of what each adjustment is intended to achieve. It emphasizes consensual, data-quality-based development of values for specific AFs rather than defaulting to arbitrary factors. It clearly discriminates between "best estimates" of the values of individual factors and adjustment for overall uncertainty, including the uncertainty associated with the AFs themselves.

The plant TRV values used for aluminum, arsenic, barium, beryllium, chromium, cobalt, copper, lead, magnesium, manganese, mercury, thallium, and vanadium were taken directly from Suter et al., (1993) and no AF values were assigned. The values presented in that paper are toxicological benchmarks for screening potential contaminants of concern for effects on terrestrial plants in soil. These values are for those contaminants potentially associated with DOE sites and were, therefore, appropriately used in the calculations for the INEEL.

Selecting QCEs—TRV development is initiated by reviewing the available toxicological literature and relevant databases for each contaminant and functional group members to identify QCEs from the best available study. Studies considering nonlethal endpoints and reporting NOAELs are selected, if available. Those reflecting reproductive competence are most preferred as such endpoints are considered to best reflect the population-level impacts of greatest concern in ERA. The following criteria are used to select QCEs:

- Experimental taxa should be as similar as possible to receptors at any applicable INEEL site(s), both physiologically and ecologically. For body size, feeding, and behavioral habits, anatomy, and physiology, the surrogate species should be matched as closely as possible to the receptors.

- Test exposure route and medium should be similar to that expected for receptors in the field. For most of the receptors at the INEEL, exposure media are limited to soil and dietary items (both animal and vegetable). Liquid intake is largely in the form of metabolic water. Dietary laboratory studies are, therefore, the most appropriate models for extrapolation. Gavage and drinking water studies will be considered if necessary, but reduce confidence in the applicability of the study.

- Long-term (preferably lifetime) exposures should be used, because they are closest to exposure patterns occurring in the field.

- Experimental endpoints should represent ecologically significant effects at the population level. In general, the loss of a few individuals of a species is unlikely to significantly diminish the viability of the population or disrupt the community or ecosystem of which the species is a part. As a result, the fundamental unit for ERA is generally the population rather than the individual, with the exception of T/E species (EPA 1992). In general, the most appropriate endpoints for ERA are reproduction, neurological function, and growth and development. For species under regulatory protection, TRVs are based on the most sensitive nonlethal endpoints referencing specifically to individuals.

- Doses within the NOAEL-LOAEL bracket should be identified. If these data are not available, the following dose levels (in decreasing order of preference) may be used: chronic-nonlethal-adverse-effect-level > no-effect-level > frank-effect-level (including lethality). The definition of adversity requires considerable analysis of the potential ecological significance of the effects reported. For example, elevated liver weight or enzyme induction could represent an adaptive response rather than toxic injury.

- Studies should be of high quality, which is defined as complete in design with adequate numbers of subjects and dose levels, lifetime duration, explicit analysis of experimental uncertainty, clear results, and well-justified conclusions.

If a single study cannot be selected (e.g., where only acute exposure, lethal endpoint studies are available), then an average of several studies of similar quality using the same or closely similar species may be used. In averaging, extreme outliers (defined as greater than two standard deviations away from the mean) are excluded. Where similar endpoints are observed in more than one study of similar quality, the lowest QCE should be used.

Information on the toxicological effects on mammalian receptors of the following contaminants was not located. Therefore, they could not be evaluated for potential risk.

- Calcium
- Iron
- Potassium

Information on the toxicological effects on avian receptors of the following contaminants was not located; therefore, these contaminants could not be evaluated for potential risk:

- • Aroclor-1260
- Antimony
- Barium
- Beryllium
- Calcium
- Chloride
- Chromium VI
- Iron
- Magnesium
- Potassium
- Silver
- Sodium.

Developing AFs—Six AFs for extrapolation from experimental studies to field exposures at INEEL are defined as follows:

I = intrataxon variability

R = intertaxon variability

Q_1 = risk assessor's certainty that the COPC actually causes the critical effect in the receptor, and that it is an ecologically significant effect

Q_2 = extrapolation from short- to long-term exposure durations

Q_3 = extrapolation across endpoint types to estimate an NOAEL

U = any residual uncertainty in the data evaluation process and estimation of other AFs based on data quality, study design, and known but otherwise unaccounted for extrapolation issues

M = correction of differences in metal bioavailability between QCE studies where soluble salts are administered via drinking water and INEEL exposure conditions (i.e., metal species are encountered in soil and dietary items)

Values for these AFs are set based on the quality of the selected study in particular, and of the database in general. Other potentially influential factors include the ecological circumstances of the receptor, regulatory criteria and standards, background contaminants levels, and protection status. To prevent needless overestimation of risk, the maximal AF product (all AFs multiplied together) is scaled to the overall extrapolation error observed in experimental studies designed specifically to determine the uncertainty in such extrapolations. Barnthouse et al., (1990) quantified the range of maximal uncertainty necessary to permit extrapolation of various kinds of toxicity data for various taxa of finfish at the population level. The types of toxicity data used included studies involving particular species of interest and other species, for acute, partial life-cycle, and full life-cycle exposures. The range of maximal uncertainty varied with the type of data used, and ranged from approximately 200 to 400 (Barnthouse et al., 1990). It is assumed that the degree of variability observed among fish taxa is similar to that occurring among other vertebrate taxa.

Based on a systematic review of all available information (Ludwig et al., 1994), a simple, relative scale is developed consisting of "low," "medium," and "high" rankings for each AF, with adjustments made of the basis of specific inherent uncertainty or variability in the particular extrapolations. The quantitative valuation of this scale is designed to be constrained by an upper bound in the range of 200 to 400, and use the most plausible values for each AF.

Specific values for these AFs and a brief description of criteria for their use are presented in Table 6-18. Values for all AFs except Q_1 and M are set at 1 ("low"), 2 ("medium"), and 3 ("high"), with lower values generally representing greater confidence that the QCEs correspond well with "safe" doses for receptors. The factor Q_1, which expresses the degree of certainty that the experimental effect will not occur in the field or is not of ecological significance, runs on a positive scale equivalent where 0.1 represents high certainty that the effect either does not occur in the receptor or is ecologically irrelevant, 0.5 represents moderate certainty that the effect does not occur or is irrelevant, and 1 represents reasonable certainty that the effect will occur in the receptor species and is ecologically significant. The medium of exposure factor M is set at 1 if the medium of exposure in the QCE study is similar to field exposure media at this site (i.e., primarily food and soil ingestion). However, because a number of toxicological studies for metals used soluble salts in drinking water as a means of exposure, and both the contaminant species and exposure matrix tend to maximize metal absorption (e.g., Steele et al., 1990; Griffin and Turck, 1991; Witmer et al., 1991), M is set at 0.5 to conservatively represent the significantly lower bioavailability of the metal species

Table 6-18. AF values and criteria for their use in developing TRVs for INEEL.

Adjustment Factor	Qualitative Ranking	Value	Criteria
I	Low	1	Variability is low
	Medium	2	Variability is moderate or average
	High	3	Variability is high, or information on variability is inadequate
R	Low	1	Test organism and functional group, T/E, and C2 species are in same taxonomic order and trophic category
	Medium	2	Test organism and functional group, T/E, and C2 species are in same trophic category but may be in different taxonomic order
	High	3	Test organism and functional group, T/E, and C2 species are in different trophic categories and taxonomic order
Q_1	Low	0.1	Experimental endpoint is highly unlikely to occur in the field
	Medium	0.5	Experimental endpoint is moderately unlikely to occur in the field
	High	1	Experimental endpoint is likely to occur in the field
Q_2	Low	1	Study was of chronic duration
	Medium	2	Study was of subchronic duration
	High	3	Study was of acute duration
Q_3	Low	1	NOAEL
	Medium	2	LOAEL
	High	3	Adverse-effect level or frank-effect level
U	Low	1	High quality studies
	Medium	2	Studies of reasonable quality
	High	3	Studies with flawed design or incomplete information
M	—	0.5	Soluble metal salt administered in drinking water
	—	1	Exposure medium comparable to those at the INEEL

associated with soils and dietary items in the natural environment. Thus, the maximum product of the seven AFs is 243. This AF maximum represents the extent to which valid extrapolation of the data can be applied across experimental protocols or among taxa. More detailed information on the definition and valuation of these factors is available from the Rocky Mountain TRV study (Ludwig et al. 1994).

Developing TRVs—The third element in ecological effects assessment is the derivation of TRVs. TRVs were derived for each functional group by selecting the experimental study with the most appropriate QCE for that chemical and assigning numeric values for all AFs to account for uncertainties associated with extrapolation across species and exposure conditions.

The algorithm used for deriving a TRV is:

$$TRV = \frac{QCE}{AF} \quad (6\text{-}31)$$

where

QCE = quantified critical exposure level

AF = $[I] \times [R] \times [Q_1] \times [Q_2] \times [Q_3] \times [U] \times [M]$.

Information used to derive TRVs for nonradioactive inorganic and organic contaminants is summarized in this section. A summary of TRVs for each contaminant/functional group/sensitive species combination is presented in Appendix G for mammalian and avian receptors. Table G-2 summarizes the TRVs for mammalian functional groups and unsensitive species. A summary of the TRVs for avian functional groups is contained in Table G-3. Shading in Tables G-2 and G-3 corresponds to the TRVs chosen for each functional group. Using the most appropriate study, when the test organism and the receptor were in the same taxonomic order and trophic category (R = 1), the corresponding TRV was chosen, as shown in heavier shading. When the test organism and the functional group are in the same trophic category an R=2 AF is used. Otherwise, the most appropriate TRV developed using R=3 was used. Little information was found describing the effects of COPCs on reptilian, invertebrate, or terrestrial plant receptors. When available, that information is summarized in Sections 6.3.4 and 6.3.5. Development of TRVs for radionuclides is described in Section 6.3.6.

6.3.4 Development of TRVS for Inorganic Contaminants of Potential Concern

This section contains summaries of the information used in determining the TRVs for the inorganic contaminants for which toxicological studies were located as follows:

- Aluminum
- Antimony
- Arsenic
- Barium
- Beryllium
- Cadmium
- Chloride
- Chromium
- Cobalt
- Copper
- Cyanide
- Fluoride
- Lead
- Magnesium
- Manganese
- Mercury
- Nickel
- Selenium
- Silver
- Sodium
- Sulfate
- Thallium
- Vanadium
- Zinc.

The development of TRVs for the studies identified for each COPC is contained in Appendix G.

Aluminum. Many of the inorganic contaminants are metals. Soils represent the most concentrated source of metals in the terrestrial environment. The health risks posed by trace metals in soils are not determined solely by their quantity. A number of contaminant, environmental, and biological conditions and processes influence the accessibility and availability of metals to organisms, and hence their toxicological significance. First, speciation is a major determinant of the fate, bioavailability, absorption, and toxicologic characteristics of metal compounds. Second, the distribution coefficient between soil and water (K_d) depends upon both the properties of the metal and the composition of the soil. This coefficient also governs the bioavailability of a metal to organisms contacting the soil, with weakly bound metals highly bioavailable and more strongly bound metals less bioavailable. Other influential factors include: (1) the characteristics of the interface (e.g., lung, skin, intestine), (2) the reactivity of the metal with the interface, and (3) the concurrent presence of other metals or other substances that may stimulate or inhibit metal uptake. There is no evidence that aluminum is essential to plants. Sensitivity to aluminum varies widely and some plants may be harmed by low concentrations of the element (USEPA), 1983). Very sensitive plants (e.g., barley and timothy) have depressed growth at soil concentrations of 2 parts per million (ppm) and tolerant plants (e.g., corn) are depressed at 14 ppm (USEPA, 1983, McKee and Wolf, 1963). There are some accumulator plants (e.g., club moss, hickory, and ash) that can tolerate large amounts of aluminum. Aluminum taken up by plants tends to accumulate in the roots and is not easily translocated to the shoots. No other phytotoxic information could be found in the technical literature.

There are no reports in the technical literature of mammal toxicity attributed to naturally occurring aluminum in the environment. It seems unlikely that grazing animals could achieve toxic concentrations naturally (Gough et al., 1979). An acute toxic dietary concentration of 20,000 mg/kg for the rat was reported by Gough et al. (1979). NAS/NAE (1972) reported that a level of 4,000 mg/kg of aluminum in the diet of chicks caused phosphorus deficiency. USEPA (1983) reports a phytotoxic concentration for vegetation from soluble aluminum of 2 to 14 mg/kg.

There are no reports in the technical literature of mammal toxicity attributed to naturally occurring aluminum in the environment. Aluminum ingested by mammals is readily eliminated by the kidneys and does not bioaccumulate or biomagnify. Studies with laboratory rats indicated that tissue concentrations of aluminum between treated and control animals were similar 7 days after withdrawal of dietary aluminum. An acute toxic dietary concentration of 20,000 mg/kg for the rat was reported by Gough et al. (1979). NAS/NAE (1972) reported that a level of 4,000 mg/kg of aluminum in the diet of chicks caused phosphorus deficiency. There is no indication from several studies that aluminum alters the reproductive capabilities of rats or mice (Clement Associated, Inc., 1990). McKee and Wolf (1963) and Underwood (1971) report that aluminum is not highly toxic to wildlife.

It is unlikely that aluminum poses a toxicological risk to wildlife. Even though concentrations of aluminum that exceeded guidelines were reported from sediment, soil, and subsurface water media, the scientific literature indicates that wildlife species can probably bioaccumulate and regulate large concentrations of this metal without harm.

The fathead minnow is not highly sensitive to aluminum, and has a 96-hour LC_{50} value of 35,000 µg/L. In contrast, the daphnid *Ceriodaphnia dubia* is far more sensitive, and has a 48-hour LC_{50} of 1,900 µg/L. In chronic toxicity tests, *Ceriodaphnia dubia* survival is reduced at 2,400 µg/L and *Daphnia magna* survival is reduced at 1,020 µg/L, but not at 540 µg/L. Early life-stage tests with the fathead minnow showed a chronic-effect level of 3,288 µg/L (USEPA, 1988).

The range of concentrations of aluminum acutely toxic to freshwater invertebrate species is about the same as the range of concentrations toxic to fish (USEPA, 1988a).

Antimony (CAS #7440-36-0). Antimony causes a number of toxic effects in animals, including suppression of weight gain, shortened life span, and damage to liver, heart, thyroid, and kidneys. Trivalent compounds (e.g., antimony trioxide, antimony trisulfide) are about 10 times more toxic than pentavalent forms. The gastrointestinal absorption of trivalent antimony is about 15–36% (Weitz and Ober, 1965; VanBruwaene et al., 1981; Gerber et al., 1982). The acute toxicity of antimony trioxide is low, with an oral LD_{50} in rats of greater than 20 g/kg (Smyth and Carpenter, 1948).

In chronic studies, 5 mg/L potassium antimony tartrate (approximately 0.35 mg/kg-day) in drinking water is associated with slightly decreased life spans in rats (Schroeder et al., 1970) and female mice (Schroeder et al., 1968; Kanisawa and Schroeder, 1969). Endpoints examined in these chronic (lifetime) studies included growth and body weight, median life span, longevity, tumor incidence, and histopathology. Other ecologically relevant endpoints (e.g., reproduction) were not examined, and only one dose was administered. Although rats appeared to be more sensitive than mice in these studies, the effects reported are of questionable ecological significance.

The recommended screening benchmark concentration for phytotoxicity in soil for antimony of 5 mg/kg was used as the TRV for terrestrial plants (Suter et al., 1993). No information on the toxicological effects of antimony on avian or reptile receptors was located.

Arsenic (CAS #7440-38-2). Arsenic is a metalloid element that is widespread in all environmental media, making up about 0.0005% of the earth's crust. Arsenic is commonly present in living organisms and is constantly being oxidized, reduced, or metabolized. Many arsenic compounds are readily solubilized in soil, making them available for plant uptake or for reduction by organisms or chemical interactions. Biological uptake of arsenic results in measurable quantities of reduced or methylated arsenic forms. Arsenic occurs naturally in all environmental media. Arsenic has four valence states: -3, 0, +3, and +5. Arsines and methylarsines, which are characteristic of compounds in the -3 state, are unstable in air. Most arsenicals degrade to yield arsenate, although arsenate may form under anaerobic conditions. Biotransformation of these compounds may occur and yield volatile arsenicals. The dominant form of arsenic present in aerobic soils is As^{+5}, while As^{+3} is the primary species in anaerobic soils. Inorganic arsenic is more mobile than organic arsenicals and thus is more likely to leach into surface or groundwaters. Trivalent species are generally more toxic, more soluble, and more mobile than pentavalent forms. Soil microbes can metabolize arsenic to volatile arsine forms. The half-life of arsenic in soil is estimated to be 6.5 years for arsenic trioxide to 16 years for lead arsenate. Soils with high organic matter content, low pH, low phosphate, and low mineral content readily sorb arsenates. In air, most arsenic particulate contains inorganic arsenic compounds, particularly As^{+3} compounds (Eisler 1988a).

At relatively low levels, arsenic stimulates growth and development in several plant species (Eisler 1988a). The bioavailability of arsenic depends on several factors including pH, soil texture, fertility level, and plant species. Inorganic arsenate is readily taken up by plants via the phosphate carrier mechanism. Therefore, plants tend to have a poor ability to distinguish arsenate from phosphate. In general, arsenic is most available to plants grown in coarse soils having little colloidal material and a low ion-exchange capacity. Conversely, fine soils high in clay, organic matter, iron, calcium, and phosphate tend to retard the bioavailability of arsenic to plants (NRCC 1978). The accumulation of arsenic in plants tends to be directly correlated with the amount of arsenic in the dissolved fraction versus total arsenic concentrations (NRCC 1978).

The potential toxicity of arsenic to any organism is dependent on its chemical form. Inorganic arsenicals are generally more toxic than organic arsenicals, and trivalent forms are more toxic than pentavalent forms. Toxicity is related to aqueous solubility, and the order of toxicity (from greatest to least) is arsines > inorganic arsenites > organic trivalent compounds > inorganic arsenates > organic pentavalent compounds > arsonium compounds > elemental arsenic (Eisler 1988a).

Chemical properties contributing to arsenic's toxicity include its ability to bind to protein sulfhydryl groups and to substitute for phosphorus in some biochemical reactions. These chemical properties may also be responsible for arsenic's apparent essentially in several mammalian species (e.g., Frost 1983; Uthus 1992). In fact, arsenical feed additives are used to promote growth in a number of agricultural species (Eisler 1988a). Recent studies have suggested that arsenic has a physiological role in the formation of various metabolites of methionine metabolism (Uthus 1992). The arsenic requirement for growing chicks and rats is approximately 25 mg/kg diet (Uthus 1992). Species differences in the pharmacokinetic disposition of arsenic have significant effects on their sensitivity to its toxic effects. In addition, animals exposed to sublethal levels of arsenic can develop tolerance to subsequent exposures (Eisler 1988a).

A subacute study using domestic sheep was documented (Eisler 1988a) in which an NOEL endpoint using 2.3 mg/kg-day was reported. An LOAEL of 1.5 mg/kg-day was reported in a chronic study using sodium arsenate in rats (Byron et al. 1967). The data did not show a good dose-response curve in the low-dose range. This study was used in the development of TRVs for rats.

The National Academy of Sciences reported a LD_{50} of 39 mg/kg-day using sodium arsenite in mallards. The recommended screening benchmark concentration for phytotoxicity in soil for arsenic of 10 mg/kg was used as the TRV for terrestrial plants (Suter et al. 1993).

Barium (CAS No. 513-77-9). Barium is distributed all over the earth and occurs most frequently as barite. Barium is used as a carrier for radium: a deoxidizer for copper; lubricant for anode rotors in x-ray tubes; in paints, soap, paper, and rubber; in the manufacture of ceramics and glass; and as a heat stabilizer for plastics. Barium metal in the free state does not occur in nature. It is found in zinc or iron ores. It is emitted mostly by industrial processes involved in the mining, refining, and production of barium and barium-based contaminants and as a result of the combustion of coal and oil. Barium is taken up, retained, and excreted in mammals in much the same way as calcium compounds.

Little information regarding the toxicity of barium is available. Its acute toxicity is low, with LD_{50}s in experimental animals consistently greater than 100 mg/kg (ATSDR 1992a). High barium concentrations (2 to 10 ppm) in human drinking water have been reported to be associated with elevated cardiovascular mortality, hypertension, and other cardiovascular effects (ATSDR 1992a).

Increased blood pressure, depressed cardiac contractility and conduction, and lower cardiac ATP content were observed in rats chronically exposed to 10¾100 mg Ba/L in drinking water (Perry et al. 1983, 1985, 1989; Kopp et al. 1985). The NOAEL exposure level identified in these studies was 1 mg/L, or approximately 0.5 mg/kg/day. A TRV for barium in carnivores was derived from an acute lethality study m which the LD_{100} was 59 mg/kg (Venugopal and Luckey 1978).

No information on the toxicological effects of barium on avian, reptile, or invertebrate receptors was located. The recommended screening level toxicological benchmark of 500 mg/kg for barium was used as the TRV (Suter et al. 1993).

Beryllium (CAS #7440-41-7). Beryllium is a metal with a complicated coordination chemistry. It can form complexes, oxycarboxylates, and chelates with a variety of materials. Although little information concerning adsorption of beryllium is available, based on its geochemical similarity to aluminum, it is expected to be adsorbed onto clay mineral surfaces at low pH levels and to be complexed into some insoluble compounds at high pH. In most natural environments, beryllium is likely to be present in sorbed or precipitated, rather than dissolved, form (EPA 1985).

Limited toxicity data is available for oral exposure to beryllium. Decreases in growth have been reported for rats exposed to beryllium. However, significant changes in organs and other physiochemical changes were not reported. Data on the teratogenicity or reproductive effects of beryllium are limited.

No adverse effects were observed in a rat chronic oral study at 5 mg/L beryllium in drinking water (Schroeder and Mitchner 1975). This study was used as the basis for deriving a TRV of 3.57 mg/kg/day.

Beryllium has been reported to produce embryolethality and terata in chick embryos (Puzanova et al. 1978) when administered subgerminally. However, no dietary toxicity studies from beryllium exposure to birds was located and a TRV was not derived.

The recommended screening level toxicological benchmark of 10 mg/kg for beryllium was used as the TRV for terrestrial plants (Suter et al. 1993).

Cadmium (CAS No. 7440-43-9). Cadmium is found naturally in the environment due to chemical weathering of rocks. It is generally found in soil as the free cadmium compounds (ATSDR 1993). There is no evidence that cadmium is biologically essential (Eisler 1985a). Cadmium is not reduced or methylated by microorganisms (ATSDR 1993). Birds and mammals are comparatively resistant to cadmium toxicity as compared to aquatic species. Sublethal effects of cadmium include growth retardation, anemia, and testicular damage (Hammons wt al. 1978) as cited in Eisler (1985a). Cadmium readily reacts with sulfhydral groups and may inhibit enzymatic reactions (Eisler 1985a). Bioaccumulation of cadmium has been reported in aquatic systems, however, only lower trophic levels are reported to exhibit biomagnification (Eisler 1985a). Accumulation of cadmium in avian species has been reported in liver and kidneys.

TRVs were developed using a multigeneration rat reproduction study by Wills et al (1981) in which a LOAEL of 5 mg/kg-day was established.

Chickens exposed to cadmium in the diet had reduced growth rates in a study by Pritzl et al. (1974). This study was used to derive a TRV for avian receptors. Behavioral changes were observed in young American black ducks when parents were fed 4 ppm cadmium for 4 months before egglaying (Heinz and Haseltine 1983; as cited in Eisler 1985a).

For invertebrates, a study on the toxicity of cadmium nitrate to the isopod (*Porcellio scaber*) was used to develop a TRV. The study reports a critical concentration of 100 µg/g cadmium in food on a dry weight basis for reproduction (Hopkin and Hames 1984).

The recommended screening level toxicological benchmark for phytotoxicity in soil of 2 mg/kg for cadmium was used as the TRV (Suter et al. 1993).

No information on the toxicological effects of cadmium on reptilian receptors was located.

Chromium (CAS No. 7440-47-31). Chromium is a multivalent element and can exist in the +2, +3, and +6 oxidation states. The latter two, chromium (III) and chromium (VI), are the most stable in the environment. In soils and sediments, chromium is influenced by oxidation and reduction reactions and can be adsorbed on the mineral and organic exchange complex or exist as a coating in iron and manganese hydrous oxide particles. Moreover, chromium may remain in solution in the pore water phase, or may become chelated by an organic liquid or precipitated (Adriano 1986; Callahan et al. 1979). The sorption of chromium (VI) by hydrous metals oxides and other soil mineral components decreases as pH levels increase. The presence of other anions (e.g., sulfate and phosphate) significantly affects the extent of adsorption by competing for adsorption sites. Formation of ion pairs, such as dissolved calcium chromate, may also reduce the extent of adsorption. In contrast to chromium (VI), the sorption of chromium (III) increases as pH units increase. In general, it appears from laboratory studies that chromium (III) is adsorbed more strongly than chromium (VI). Organic material may also be an important adsorbent in sediments and soils. Slight enrichment of chromium occurs in the humic fraction. Typically, in normal, well-drained soils, the great majority of chromium is in the form of chromium (III).

Chromium (VI) is generally more toxic than chromium (III). Although most chromium (VI) is reduced to chromium (III) in the acidic environment of the stomach (Donaldson and Barreras 1966), chromium (VI) compounds are absorbed significantly more efficiently from the gastrointestinal tract (2 to 10% of administered dose) than chromium (III) compounds (Outridge and Scheuhammer 1993). Once absorbed, chromium (VI) is quickly reduced to the trivalent form. The damaging effects of chromium (VI) are caused by its greater membrane permeability, which allows it to cross biological membranes and oxidize cellular components not normally accessible to chromium (VI). As a result, the differences in systemic toxicity are primarily attributable to differential solubilities and absorption rates of the two valence states (Franchini and Mutti 1988).

The mobility of chromium (VI) and the limited supply of extracellular reductants causes chromium (VI) to be distributed more widely in the body than chromium (III). The intracellular reduction of chromium (VI) to chromium (III) generates unstable intermediate chromium (V) and chromium (IV) ions, active oxygen species (hydroxyl and superoxide radicals, single oxygen), and thiyl and organic radicals that are responsible for the cytotoxicity, mutagenicity, and carcinogenicity of the hexavalent form (reviewed by Manzo et al. 1992; Cohen et al. 1993; O'Flaherty 1993; Outridge and Scheuhammer 1993).

Chromium exhibits a pattern of biominification rather than biomagnification in ecological food webs. Because the speciation of chromium (VI) taken up by plants is poorly understood, it is assumed to be the primary form of exposure to herbivores. However, chromium (VI) is immediately converted to chromium (III) in animal tissues. Therefore, carnivorous receptors will be primarily exposed to the less toxic trivalent form. Development of TRVs based on chromium (VI) for receptors higher in the food chain is thus highly conservative, and will tend to overestimate chromium-related risk to these receptors.

In a study of chromium toxicity (Rosomer et al. 1961), subchronic NOAEL of 100 mg/kg in the diet for chickens were reported. This information is used to estimate the TRV for avian functional groups.

Pregnant female mice receiving 250 mg/L potassium dichromate in drinking water throughout gestation showed no clinical signs of toxicity, but produced significantly fewer viable offspring (Trivedi et al. 1989). In the dog, 6 mg/L in drinking water (approximately 0.3 mg/kg/day) was a chronic NOAEL [Steven et al. 1976 (cited in Eisler 1986)]. A similar level was without observable effects in a study of chronic toxicity (Anwar et al. 1961). Based on these results, TRVs were derived for mammalian functional groups.

Cobalt. IRIS contains no toxicity information on cobalt. Cobalt is a rare metal produced mostly as a by-product of copper smelting. It is an essential nutrient found in vitamin B-12. A B-12 deficiency in humans results in pernicious anemia.

Environmental exposures to high levels of cobalt rarely occur. Industrial exposures to airborne cobalt are known to cause respiratory irritation, perhaps as the result of sensitization. Ingestion of high levels of cobalt usually occur as a result of medical therapy. Excess cobalt can cause goiter, polycythemia, and possible cardiomyopathy.

Copper (CAS 7440-50-8). Copper is one of the least mobile of the trace elements and tends to be uniformly distributed in the soil horizon. Soil parameters that influence copper availability include pH, CEC, and organic matter content. Persistence of copper in soils is caused by binding to organic matter, the formation of oxides with iron and manganese, the presence of clay minerals, and soil pH. A pH of 6 or less increases the mobility and availability of copper in soil. Copper is one of the trace elements most extensively complexed by humic materials. Most copper is readily available to plants when the soil pH is below 6, especially in soils with low organic matter and humic material content. Sulfides, which may prevail in soils under reducing conditions, effectively precipitate copper, thereby reducing the bioavailable amount of copper. Biogenic ligands bind with copper, resulting in the precipitation and sorption of copper. Copper is one of seven essential plant micronutrients. Copper in soil tends to strongly bind with organic matter, which limits it's availability for uptake by plants.

Copper is widely distributed in nature and is an essential element for (1) the normal function of several critical enzymes and (2) the utilization of iron. Copper deficiency is, therefore, usually a greater health concern than copper excess. Copper absorption in the gastrointestinal tract is normally regulated by body stores. Absorbed copper is transported to the liver, where it may be incorporated into ceruloplasmin (a copper transport and donor molecule) and excreted into the plasma, stored as metallothionein or in lysosomes, or excreted via the bile (reviewed by Nederbragt et al. 1984).

Depressed food intake, body-weight gain, egg number and weight, and organ weights are associated with copper excess in poultry (Stevenson and Jackson 1981). The pair-feeding study was conducted to determine whether these effects were associated with direct toxicity or the accompanying marked reduction in food intake (Stevenson and Jackson 1981). Body weight, food intake, organ weights; egg production; egg weight; clinical chemistry parameters; and organ copper, iron, and zinc concentrations were monitored in laying hens fed varying concentrations of copper in their diet for 6 weeks (Stevenson and Jackson 1981). A NOAEL of 24 mg/kg/day was identified and used to develop TRVs for avian functional groups.

High doses of copper have caused liver and kidney damage as well as anemia in a number of species. It has been observed that the stomach is also a target in rats and mice (Hebert et al. 1993). This well-designed subchronic feeding study examined histopathology, clinical pathology, reproductive toxicity, and tissue metal accumulation in males and females of both species. A QCE of 66 mg/kg/day (NOAEL) was identified from this study and used to develop mammalian TRVs.

A mammalian TRV was also derived from a chronic feeding study in mink (Aulerich et al. 1982). The purpose of this study was to determine whether copper supplements would improve growth and survival. Endpoints examined included the effects on growth, blood chemistry, reproductive performance, and kit survival and development. The QCE from this study is a NOAEL of 12.9 mg/kg/day.

An oral NOAEL was established in a chronic study of young calves (Cunningham 1946). The study confirms that young calves are susceptible to copper. The QCE from this study is 1.1 mg/kg-day.

The recommended screening benchmark concentration for phytotoxicity in soil for copper of 40 mg/kg was used as the TRV for terrestrial plants (Suter et al. 1993).

Cyanide. Many chemical forms of cyanide are present in the environment (e.g., metallocyanide, synthetic organocyanides, free CN). Only free cyanide is the primary toxic agent. Cyanides are readily absorbed through multiple pathways (inhalation, ingestion, dermal) and are rapidly distributed through the body. However, there are no reports of cyanide biomagnification or cycling in living systems. Cyanide is a potent and rapid asphyxiant. However, diagnosis of acute cyanide poisoning is often difficult because signs and symptoms are nonspecific and toxicity is influenced by a variety of factors. Deficiencies of vitamin B12, iodine, and sulfur amino acids can modify its biocidal properties.

A rat chronic oral toxicity study (Howard and Hanzal, 1955; EPA, 1984) indicated a NOAEL of 20.4 mg/kg-day that was used to generate a TRV for mammalian receptors.

Cyanides have been used as feeding stimulants for some insects (Eisler, 1991). Instar larvae of the armyworm prefer cyanogenic foods, such as foliage of the lima bean. However, data are scarce for other invertebrates. As such, a cyanide TRV was not developed for invertebrates.

No information on the toxicological effects of cyanide on reptile receptors was located.

Fluoride (CAS No. 16984-48-8). Inorganic fluorides are generally highly irritating and toxic. Acute effects resulting from exposure to fluorine compounds are due to HF. Chronic fluorine poisoning, or "fluorosis," occurs among numbers of cryolite, and consists of a sclerosis of the bones caused by a fixation of the calcium by the fluorine. There may also be some calcification of the ligaments. The teeth are mottled, and there is osteosclerosis and osteomalacia. Large doses can cause very severe nausea, vomiting, diarrhea, abdominal burning and cramp-like pains. Fluoride is not taken up by the thyroid and does not interfere with iodine uptake. It can cause or aggravate attacks of asthma and severe bone changes, making normal movements painful. Some signs of pulmonary fibrosis have been noted (Sax and Lewis 1987).

The reproductive effects of fluoride administered orally in the diet of minks was studied (Aulerich et al 1987). Five dose levels were administered. Fluoride up to 229 ppm had no adverse effects on reproduction. Survivorship of kits in the 385 ppm group was significantly reduced. These doses were considered to be NOAELs and LOAELS, respectively. Because the study considered exposure over 382 days including critical life stages (reproduction), these doses were considered to be chronic. A NOAEL of 31.37 mg/kg/d was established.

The effects of fluoride administered to the screech owl orally in the diet for a period of 5 to 6 months were studied (Pattee et al. 1988). The fertility and hatching success were significantly reduced by 232 ppm fluorine in the diet, 56.5 ppm fluorine in the diet had no adverse effect. Because the study considered exposure during reproduction, these doses were considered to be chronic. A NOAEL of 7.8 mg/kg/d was established.

Lead (CAS No. 7439-92-1). Lead is a ubiquitous trace constituent in rocks, soils, plants, water, and air, with an average concentration of 16 mg/kg in the earth's crust (Eisler 1988b). Lead has four stable isotopes: Pb-204 (1.5%), Pb-206 (23.6%), Pb-207 (22.6%), and Pb-208 (52.3%). Lead occurs in four valence states: elemental (Pb°), monovalent (Pb^{+}), divalent (Pb^{+2}), and tetravalent (Pb^{+4}). In nature, lead occurs mainly as Pb^{+2} and is oxidized to Pb^{+4}. Metallic lead is relatively insoluble in hard waters. Some lead salts are somewhat soluble in water. Of the organoleads, tetraethyllead and tetramethyllead are the most stable and are highly soluble in many organic solvents but are fairly insoluble in water. Both undergo

photochemical degradation in the atmosphere to elemental lead and free organic radicals. Organolead compounds are primarily anthropogenically-produced (Eisler 1988b).

Lead is neither essential nor beneficial to living organisms. Lead affects the kidney, blood, bone, and central nervous system. Effects of lead on the nervous system is both functional and structural. Lead toxicity varies widely with the form and dose of administered lead. In general, organolead compounds are more toxic than inorganic lead. In nature, lead occurs mainly as divalent, Pb^{2+}. Ingestion of lead shot by regulatory waterfowl is a significant cause of mortality in these species.

Hatchlings of chickens, quail, and pheasants are relatively tolerant to moderate lead exposure (Eisler 1988b). There was no effect on hatchling growth of these species at dietary levels of 500 mg/kg or on survival to 2,000 mg/kg lead (Hoffman et al. 1985 as cited in Eisler 1988). For avian herbivores, a TRV was estimated using a study of mallards (Dieter and Finley 1978). Altricial species are generally more sensitive to lead than precocial species (Eisler 1988b) of avian insectivores. An oral study using European starlings (Osborn et al. 1983) was used to generate a TRV for trimethyllead chloride. Because organic lead compounds are generally more toxic than inorganic lead, the TQs generated using this TRV should be interpreted with caution. American kestrel (*Falco sparverius*) exposed to 50 mg/kg/day metallic lead in diets did not exhibit effects on survival or reproductive success (Colle et al. 1980). Using these studies, TRVs were developed for avian functional groups.

Studies using rats administered lead in drinking water (Kimmel et al. 1980), of lead toxicity in calves (Zmudzki et al. 1983), and using dogs (Demayo et al. 1982) were used to develop TRVs for mammalian receptors.

A study on the toxicity of lead nitrate to the isopod (*Porcellio scaber*) reports a critical concentration of 2,000 g/g lead in food on a dry weight basis for reproduction (Hopkin and Hames 1994).

The recommended screening benchmark concentration for phytotoxicity in soil for lead of 50 mg/kg was used as the TRV for terrestrial plants (Suter et al. 1993).

Manganese (CAS #7439-96-5). The transport and partitioning of manganese are influenced by the solubility of the particular form present, which, in turn, is determined primarily by the pH oxidation and reduction potential. Manganese may exist in one of four oxidation states: 2+, 3+, 4+, and 7+. Divalent manganese (Mn^{+2}) exists mostly in waters with a pH of 4 to 7. The likelihood that soluble manganese compounds will sorb to soils is affected primarily by the CEC and the organic matter content of the soil. Soil sorption can vary by as much as five orders of magnitude depending on soil conditions. The oxidation state of manganese in soil may be altered by microbial populations (ATSDR 1992b). Manganese affects the central nervous system in humans. However, it is important to recognize the substantial difference in species requirements for manganese. Toxic levels of manganese in humans do not meet the nutritional requirements of rats (IRIS 1994).

The bioavailability of different forms of manganese varies considerably depending on different exposure conditions. There is potentially higher bioavailability of manganese from drinking water than food. It is also important to recognize that various dietary factors as well as the form of manganese can have a significant bearing on the dose absorbed from the gastrointestinal tract. For instance, many constituents of a vegetarian diet (e.g., tannins, oxalates, phylates, fiber, calcium, and phosphorus) have been found to inhibit manganese absorption presumably by forming insoluble complexes in the gut. Thus, herbivores are more likely to be resistant to manganese toxicity. Also, the form of manganese can significantly influence toxicity. For example, mice receiving the two soluble forms of manganese (chloride

and acetate salts) were found to gain significantly less weight than controls, while mice consuming the insoluble forms of manganese (carbonate and dioxide salts) appeared to actually gain slightly more weight than controls.

The manganese requirements vary considerably between species. In terms of dietary concentration (ppm), the requirements of young animals have been estimated as follows:

- dog, 4.5
- rabbit, 8.5
- pig, 4
- calf, 40
- sheep, 30
- rat, 50
- chick, 55
- turkey, 55 (NAS 1980).

A study reporting the minimum manganese requirements in chickens was used to derive a TRV of 2.9 mg/kg/day. Guinea fowl were found to have reduced hatchability and increased deformed embryos when fed diets deficient in manganese (Offiong and Abed 1980). A dietary reproduction study in rats exposed to 250 ppm manganese (13 mg/kg/day) was used to develop a TRV of 1.1 mg/kg/day (Laskey et al. 1982).

No information on the toxicity of manganese to reptiles or invertebrates was located. The recommended screening benchmark concentration for phytotoxicity in soil for manganese of 500 mg/kg was used as the TRV for terrestrial plants (Suter et al. 1993).

Mercury (CAS No. 7439-96-5). Mercury exists in the environment in three oxidation states: the element itself, +1 (mercurous) state, and +2 (mercuric) state. The factors that affect which species dominates in an environment are the redox potential and the pH of the system. Particle-bound mercury can be converted to insoluble mercury sulfide, which can be bioconverted into more soluble or volatile forms that may reenter the atmosphere or be taken up by biota and bioaccumulated in the terrestrial food chain. Mercury forms many stable organic complexes that generally are more soluble in organic matter than in water. Inorganic and organic particles strongly sorb mercury. Mercury can be transformed in the environment by biotic and abiotic oxidation and reduction, bioconversion of organic and inorganic forms, and photolysis. Mercury can be strongly concentrated by living organisms (Callahan et al. 1979). The chemistry of mercury in the environment is complex, not only because of its various oxidation states but also because of biotic and abiotic methylation and demethylation processes, complexation with organic and inorganic ligands, and the differential solubility and volatility of various forms. As speciation is a major determinant of the fate, bioavailability, absorption, and toxicologic characteristics of mercury compounds, lack of knowledge of the state of the mercury in INEEL soils is a large source of uncertainty in both exposure assessment and TRV development.

Although the generally more toxic organic forms of mercury are unlikely to persist in the environment, they (in particular, methylmercury) may be formed in biotic tissues and are known to biomagnify through ecosystems, particularly aquatic systems (reviewed by Wren 1986; Scheuhammer 1987). Thus, to ensure that mercury TRVs for the WAG ERA are protective of receptors at all levels of ecological organization, TRVs are developed from studies investigating the toxic effects of organic mercurials. It is noted that this measure is highly conservative and will tend to overestimate risks for receptors lower in the food web because the majority of mercury in soil and plants (i.e., the majority of exposure to plants and soil-dwelling and herbivorous animals) is expected to be inorganic.

Because of its chemical stability and lipophilicity, methylmercury readily penetrates the blood-brain barrier. The central nervous system is thus a major target organ in both mammals and birds. However, reproductive effects have been reported at even lower doses. Methylmercury can be converted to inorganic mercury both in tissues and by microflora in the gut. The homolytic cleavage of the mercury-carbon bond leads to generation of reactive intermediates, e.g., methyl and metal radicals, which cause cellular damage (reviewed by Wren 1986; Scheuhammer 1987; Manzo et al. 1992).

The effects of mercury on avian herbivores, insectivores, and carnivores were evaluated as follows. For herbivores, the effects of organic mercury compounds on galliformes (domestic chickens, quail, pheasants) have been investigated by several groups. However, no study was reviewed that identified an NOAEL. The lowest LOAEL for relevant endpoints (reproductive success) of several similar studies was found in a study of the effects of mercury to birds (Finnreite 1979). Reduced egg production, shell thickness, and hatchability in pheasants fed seed treated with organomercurial fungicide were observed. This study was selected over others because of its use of a wild species and lower dose levels. A TRV was derived from this study.

Three goshawks were fed a diet of chickens that had eaten wheat dressed with an organomercurial fungicide (Borg et al. 1970). Their tissues contained 10 to 40 ppm of mercury, mostly as methylmercury. The hawks died after 30 to 47 days; their total mercury intake was about 20 mg/bird.

Two studies examined the effects of subchronic methylmercury exposure on the reproductive competence of male and female rats (Khera and Tabacova 1973; and Khera 1973). The NOAEL identified for both sexes was 0.25 mg/kg/day. Much less information is available regarding methylmercury toxicity to herbivores. In a study of acute methylmercury toxicity in mule deer (*Odocoileus hemionus hemionus*) 17.88 mg/kg was said to be the LD_{50} (Eisler 1987a). A number of studies have examined the effects of chronic methylmercury ingestion on carnivorous mammals, particularly cats (e.g., Albanus et al. 1972; Charbonneau et al. 1976; Eaton et al. 1980) and mink (e.g., Aulerich et al. 1974; Wobeser et al. 1976; Wren et al. 1987). The chronic toxicity of cats study by was considered superior to other available studies because of its long duration (2 years), use of relatively large group sizes, detailed examination of endpoints, identification of both no-effect and effect levels, and administration of mercury via both contaminated fish and addition to diet (Charbonneau et al. 1976).

A TRV of 0.3 mg/kg was assigned for mercury for terrestrial plants based on the toxicological benchmark (Suter et al. 1993).

Nickel (CAS #7440-02-0). Nickel is a naturally occurring silvery metal that is found in the earth's crust (ATSDR 1988). Organic complexing agents may restrict soil movement and availability of nickel through the formation of organo-nickel complexes (ATSDR 1988). However, nickel ferrite appears to be the most probable nickel species to precipitate in soil. . Nickel is continuously transferred between, air, water, and soil via various natural processes including weathering, runoff, erosion, and leaching. Nickel is very

persistent in both soil and water. In the atmosphere, nickel exists primarily in the aerosol form. Nickel can exist in water in various soluble and insoluble forms depending on the physicochemical properties of the water. The mobility of nickel in aqueous media is affected by complexation, precipitation and dissolution, oxidation and reduction, and adsorption and desorption. The average residence time of nickel in soil is estimated to be 2,400 to 3,500 years. Although nickel is very persistent in soil, it can leach into groundwater. The sorption of and nickel to soils correlates with pH, total iron, and organic matter content. Organic complexing agents in soil tend to restrict the movement of nickel by forming organs-nickel compounds. Nickel is fairly mobile in soils a low pH and cation exchange capacity (ATSDR 1988).

Small amounts of nickel can be essential for normal growth and reproduction (ATSDR 1988). Oral exposure to high concentrations of nickel has been reported to adversely affect the hematological system and reproduction.

Toxicity studies in chicks by Weber and Reid (1968) and in mallards (Eastin and O'Shea 1981) were used to develop TRVs for avian herbivores.

Rats fed 5 mg/kg/day nickel sulfate in a two-year dietary study did not produce hepatic changes or altered body weights (Ambrose et al. 1976). This NOAEL was supported by a rat subchronic drinking water study conducted by American Biogenics Corp. (ABC 1986) and a rat reproductive study by RTI (1987). Using the Ambrose study, a TRV for small mammals was developed. For mammalian herbivores, a subchronic study (O'Dell et al. 1979 as cited in NAS 1980) of cows that did not exhibit reduced food intake or growth rate when fed 250 mg/kg-d nickel carbonate was used to develop a TRV. A dietary study exposing dogs to 1000 ppm Ni did not result in adverse effects (Ambrose et al. 1976) and was used to develop a TRV for mammalian carnivores.

No information on the toxicological effects of nickel on reptile or invertebrate receptors was located. The recommended screening benchmark concentration for phytotoxicity in soil for nickel of 25 mg/kg was used as the TRV for terrestrial plants (Suter et al. 1993).

Selenium (CAS #7782-49-2). Selenium is a critical nutrient and a key component of several enzymes (Eisler, 1985b). It is often found in high concentrations in areas where soils have been derived from Cretaceous rocks (Eisler, 1985b). Selenium does accumulate to high concentrations in certain species of plants (e.g., *Aster*, *Astragalus*) (Eisler, 1985b). Livestock species ingesting these plants have been reported to exhibit toxic symptoms such as abnormal movements, labored breathing, dilated pupils, bloating, diarrhea, and rapid pulse. No effective treatment is known for counteracting the toxic effects of high levels of ingested selenium. Prolonged exposure to more moderate levels of selenium result in skin lesions involving alopecia, hoof necrosis and loss, emaciation and increased serum transaminases, and alkaline phosphatase in animals (TOXNET, 1996). Selenium has been reported to cause growth retardation, decreased fertility, embryotoxicity, fetotoxicity, and teratogenic effects in animals (TOXNET, 1996). Birds appear to be particularly susceptible to selenium, particularly in the area of reproductive success. Malformations in chickens and waterfowl have been widely reported (EPA, 1993).

Selenium deficiency is often a greater threat to health than selenium poisoning (Eisler, 1985b). Selenium deficiency has been documented in a variety of species including fish, quail, ducks, poultry, rats, dogs, domestic grazing animals, antelope, monkeys, and humans (Eisler, 1985b). Selenium can also reduce the toxicity of other heavy metals such as thallium, arsenic, and copper (Wilber, 1980).

Studies by Ort and Latshaw (1978), Heinz et al., (1987), and Smith et al., (1988) evaluating the effects of sodium selenite in chickens, mallards, and black-crowned night herons were used to derive TRVs for avian receptors.

In a study by Rosenfeld and Beath (1954), selenium administered as potassium selenate to sires and pregnant rats through five breeding cycles did not affect reproduction, the number of young reared, or on the reproduction of two successive generations of dams and sires in groups receiving 1.5 ppm selenium. Because no effect on growth in rats has been reported at concentrations of 1.6 to 4.8 ppm selenium in the diet (Halverson et al., 1966), a reproductive endpoint was selected to develop a TRV. Selenium doses of as low as 3.2 mg/kg body weight have resulted in death in sheep (Eisler, 1985b). A TRV was developed for mammalian herbivores using these data.

No information on the toxicity of selenium in reptile or invertebrate receptors was located. The recommended screening benchmark concentration for phytotoxicity in soil for selenium of 1 mg/kg was used as the TRV for terrestrial plants (Suter et al., 1993).

Silver (CAS #7440-22-4). The precious metal silver is relatively rare in the earth's crust and does not occur regularly in animal tissues. As a result, the toxicity of silver has been little studied. Approximately 1–10% of ingested silver is absorbed; as much as 18% may be retained. Silver-protein complexes accumulate in the liver, and biliary excretion (complexed with glutathione) is the major route of elimination. In most tissues, silver is deposited as large granules. With rare exceptions, these deposits are not associated with adverse effects. The LD_{50} of silver in rats is relatively high at 24 mg/kg (reviewed by Rungby, 1990).

Silver causes a conditioned deficiency of selenium in rats, decreasing tissue levels of selenium, and the selenoprotein glutathione peroxidase (Ganther, 1980). Silver ions complex strongly to sulfhydryl groups and cause peroxidation of hepatocellular membrane lipids (Rungby et al., 1987; Shinogi and Maeizumi, 1993). Because of its affinity for sulfhydryls, the degree of binding to cellular macromolecules and toxicity of silver is mitigated by induction of the divalent metal-binding protein metallothionein (Shinogi and Maeizumi, 1993). Exposure of fetal and adult rats to silver results in deposition in the central nervous system (Rungby and Danscher, 1983a, b). Pyramidal cells in the developing hippocampus appears to be a sensitive target, exhibiting reduced cellular volume in both pre- and postnatally exposed rats (Rungby et al., 1987; Rungby, 1990).

The mammalian TRVs for silver are based on a subchronic study by Rungby and Danscher, (1984) in which mice exposed to approximately 18 mg/kg/day were observed to be "hypoactive." Although silver deposits occurred in certain motor centers of the brain, no association between the concentration of deposits and the extent of hypoactivity was found.

No information on the toxicological effects of silver on avian, reptile, amphibian, or plant receptors was located. The recommended screening benchmark concentration for phytotoxicity in soil for silver of 2 mg/kg was used as the TRV for terrestrial plants (Suter et al., 1993).

Sodium (CAS #7440-66-6). Sodium is essential for all living organisms as it is important in maintenance of osmotic pressure, body fluid balance, and hydration of the tissues. Only one acute toxicity study had available data for sodium. An LD_{50} was reported for mice given 4,000 mg/kg intraperitoneally (TOXNET). No data on the toxicological effects of sodium to avian, reptilian, and plant species were available. Hence, TRVs for all avian and reptilian functional groups and plants could not be established.

Sulfate. Sulfates are generally of low toxicity. Several studies indicate no adverse effects when sulfate compounds are administered (Brown and Ganatero 1970, Sasse and Baker 1974, Paterson et al. 1979) and others that list effects of loose feces and decrease intake (Bird 1972, L'Estrange et al 1969). These five studies were conducted using pigs, chicken, pigs, and sheep. One study did list an LD_{50} for a single dose injection of sodium sulfate monohydrate in mice of 45.6 mg/kg-d (Nofre et al. 1963). To develop TRVs, studies identified for sodium sulfate were used.

Thallium (CAS #7440-28-0). Thallium is a nonvolatile heavy metal element that is not used extensively by industry and is mainly introduced into the environment as a waste product of other metals. Thallium can exist in the atmosphere as an oxide, a hydrazide, a sulfate, or a sulfide. Thallium is present in mono- or trivalent forms in the environment. Thallium (III) forms some organometallic compounds and thallium (I) forms relatively few complexes with the exception of those with halogen, oxygen, and sulfur ligands. Thallium can be removed from solution by adsorption onto clay minerals, bioaccumulation, or (in reducing environments) precipitation of the sulfide. Increased pH values have been found to produce extensive thallium-humic acid interactions while lowering thallium-inorganic interactions. Thallium may be bioconcentrated by living organisms (Callahan et al. 1979). Thallium (I) is more stable and resembles the alkali metal cations in many of its chemical properties. Thallium (III) forms many organic compounds (Zitko 1975), the toxicity of which has been little explored.

Thallium is slightly more acutely toxic to mammals than mercury. The similarity between kinetic profiles of inorganic trivalent and monovalent thallium species suggests that they are converted in vivo to one chemical form, probably monovalent thallium (Sabbioni et al. 1980). Isomorphic with potassium, thallium (I) is readily absorbed and distributed throughout the body, and can substitute for potassium and other monovalent cations in enzymatic reactions. The affinity of thallium (I) for enzymes is 10 times higher than that of potassium, which may cause the observed toxic effects (Zitko 1975). Thallium (I) uncouples oxidative phosphorylation, adversely affects protein synthesis, and inhibits a number of enzymes including alkaline phosphatase and succinic dehydrogenase (Zitko 1975). Thallium is also toxic to plants, inhibiting chlorophyll formation and seed germination.

A study in the 1930s of the acute toxicity of thallium sulfate in game birds including quail (Shaw 1933) formed the basis for the TRV for these functional groups. In a study of the acute toxicity of thallium sulfate in three immature golden eagles *(Aquila chrysaetos),* the acute oral LD_{50} was estimated to be between 60 and 120 mg/kg (Bean and Hudson 1976). Using the lower end of this range as the QCE, a TRV for raptorial birds at the INEEL was derived.

Rats exposed to thallium in their drinking water have shown effects on various neurological (Manzo et al. 1983; Rossi et al. 1988) and reproductive (Formigli et al. 1986) endpoints. Because of the clear ecological relevance of reproductive impairment, a QCE was selected from the study of thallium-induced testicular toxicity (Formigli et al. 1986).

No information on the toxicological effects of thallium on reptiles or terrestrial invertebrates was located. The recommended toxicological benchmark of 1 mg/kg for thallium was used as the TRV for terrestrial plants (Suter et al. 1993).

Vanadium (CAS #7440-62-2). Vanadium occurs naturally in igneous rock, shales, in some uranium and iron ores and in association with fossil fuels. In the environment, vanadium is usually combined with oxygen, sodium, sulfur or chloride (ATSDR 1992c). There is no indication that vanadium is nutritionally required by higher plants and annuals (Ammerman et al. 1973). Vanadium uptake into above-ground parts of terrestrial plants is low. However, some legumes have been identified as vanadium accumulators

(ATSDR 1992c). In general, bioconcentration and biomagnification in terrestrial environments appears limited.

Most toxic effects of vanadium are associated with inhalation of vanadium pentoxide (ATSDR 1992c). Vanadium is poorly absorbed in the gastrointestinal tract and most is excreted unabsorbed in feces (ATSDR 1992c).

A study of vanadium toxicity in female leghorn chickens by Kubena and Phillips (1982) was used to develop TRVs for all avian groups except for AV142 and AV143. A TRV for AV142 and AV143 was derived using a study by White and Dieter 1978.

A study of effects of vanadium to mice (Schroeder and Balassa 1967) was used to derive TRVs for mammalian species for vanadium. There is little information in the literature regarding vanadium toxicity in ruminants (Ammerman et al. 1973). A study was used to derive a TRV of 0.42 mg/kg/day (Abbey 1968).

No toxicity information on the effects of vanadium to reptiles or invertebrates was located. The recommended screening benchmark concentration for phytotoxicity in soil for vanadium of 2.5 mg/kg was used as the TRV for terrestrial plants (Suter et al. 1993).

Zinc (CAS #7440-66-6). Zinc is found naturally in the environment and is present in all foods (ATSDR, 1993b). It is an essential element and occurs in the environment in the 2+ state. Zinc is likely to be strongly sorbed to soil. Relatively little land disposed zinc is expected to be in a soluble form. Bioconcentration factors of soil zinc by terrestrial plants, invertebrates, and mammals are 0.4, 8, and 0.6, respectively (ATSDR, 1993b).

Excessive dietary zinc has been shown to cause copper deficiency and anemia (ATSDR, 1993b). Cadmium has also resulted in the redistribution of zinc to the liver and kidney. Health effects associated with zinc exposure include anemia, liver necrosis, fetal resorption, and in extreme cases, cessation of reproduction (ATSDR, 1993b).

Only one study revealed an NOAEL for chickens. A decrease in egg production was observed in chickens fed 20 mg/kg zinc sulfate in their diet. This LOAEL was converted to 12 mg/kg-day to develop a TRV (Stahl et al., 1990).

A rat developmental study by Schlicker and Cox (1968) was used to develop a TRV for small mammals. A study of sheep by Allen et al., (1983) revealed pathological changes in liver and kidney. Using these studies, a TRV was developed. A feeding study evaluating zinc oxide exposure in ferrets (Straube et al., 1980) was used to develop a TRV for mammalian carnivores. A study using dogs by Drinker et al., (1927) was used as the basis for deriving a TRV for mammalian omnivores.

A study on the toxicity of zinc nitrate to the isopod (*Porcellio scaber*) reports a critical concentration of 1,000 μg/g (mg/kg) zinc in food on a dry weight basis for reproduction (Hopkin and Hames, 1994). Since the bioconcentration factor for zinc for terrestrial plants is 0.4 and the maximum detected concentration in site soil is 2,400 mg/kg, the conservatively estimated plant concentration of 960 mg/kg falls below this critical concentration. Thus, although there is an absence of invertebrate toxicity data for soil, there is some evidence that the concentrations of zinc at the site would not result in adverse effects in invertebrates at the site.

The recommended screening benchmark concentration for phytotoxicity in soil for zinc of 20 mg/kg was used as the TRV for terrestrial plants (Suter et al., 1993). No information on the toxicological effects of zinc on reptiles was located.

6.3.5 Development of TRVs for Organic Contaminants of Potential Concern

The following section summarizes the information used in determining the TRVs for organic contaminants for which toxicological studies were located.

Dioxins/Furans. Polychlorinated dibenzodioxins (CDD)s and polychlorinated dibenofurans (CDF)s are chemically classified as halogenated aromatic hydrocarbons. They can be formed as unintentional by-products through a variety of chemical reactions and combustion processes. In general, these compounds have very low water solubility, high octanol-water partition coefficients, and low vapor pressure and tend to bioaccumulate.

The environmental fate and environmental distribution of these compounds are not yet well understood. CDDs/CDFs entering the atmosphere are removed either by photodegradation or by deposition. In soil, sediment, and the water column, CDDs/CDFs are primarily associated with particulate and organic matter because of their high lipophilicity and low water solubility. Because of their very low water solubilities and vapor pressures, CDDs/CDFs below the soil surface are strongly absorbed and show little upward or downward vertical migration. Burial in-place, resuspension back into the air, or erosion of soil to water bodies appears to be the predominant fate of CDDs/CDFs sorbed to soil. When entering the aquatic environment, most are associated with particulate matter and are likely to remain sorbed to the particulate matter once in the aquatic environment. They primarily undergo sedimentation and burial. The ultimate environmental sink of CDDs/CDFs is believed to be aquatic sediments.

These compounds exhibit little potential for significant leaching or volatilization once sorbed to particulate matter and are extremely stable under most environmental conditions. The only environmentally significant transformation process is believed to be photodegradation of nonsorbed species in the gaseous phase, at the soil-air or water-air interface, or in association with organic cosolvents.

TRV values for 2,3,7,8-Tetrachloro dibenzodioxin were used for Tetrahydrofuran in the organic screening. The following discussion is for 2,3,7,8-Tetrachloro dibenzodioxin. 2,3,7,8-Tetrachloro dibenzodioxin is a confirmed carcinogen with experimental carcinogenic, neoplastigenic, tumorigenic, and teratogenic data. One of the most toxic synthetic chemicals. A deadly experimental poison by ingestion, skin contact, and intraperitoneal routes. It is very toxic to some animals, with an LD50 of only about 0.6 μ g/kg body mass in male guinea pigs. The type and degree of its toxicity to humans in largely unknown; it is known to cause a severe skin condition called chloracne. Human systemic effects by skin contact (allergic dermatitis). Experimental reproductive effects. Human mutation data reported. Also, TCDD is a known eye irritant.

Total tetrachlorodibenzodioxins (TCDD-TOT) have a risk-based concentration of 4.0E-7 ug/L (Region III, EPA, 1995 tables of screening-level RBCs); the carcinogenic slope factor for oral ingestion of TCDD is 1.56E+5 $(mg/kg/d)^{-1}$. There is also a maximum contaminant level (MCL) of 3E-08 mg/L assigned to total TCDD-TOT. Research into health effects for tetrachlorodibenzodioxins, particularly 2,3,7,8-TCDD, is as follows.

Tetrachloridbenzo-p-dioxin has been shown to be extremely toxic to a number of animal species, the acute oral LD50 values ranging from 0.0006 to 0.283 mg/kg, the guinea pig being the most susceptible

species. However, it should be emphasized that mortality does not occur immediately, the animals undergoing a slow but progressive decline into a moribund state associated with an increased incidence of infections and the eventual death some 14 to 28 days after treatment.

Rodents exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) demonstrated severe thymus atrophy. Histologic evaluation of the thymus revealed cortical lymphoid depletion similar to cortisone-induced thymus atrophy. Depressed antibody responses, graft-versus-host, and lympho-proliferative responses were observed at slightly higher doses of TCDD. In addition, increased susceptibility to challenge with the bacteria salmonella bern was noted at low dosages. Depressed antibody responses were also observed in guinea pigs receiving cumulative dosages of TCDD as low as 0.32 μg/kg over an eight-week period. Depressed T-cell function was observed following exposure of adult mice to TCDD, which was associated with an increase in suppressor T-lymphocyte expression and loss of T-lymphocyte cytotoxicity for tumor target cells. Depressed antibody responses and depressed lymphoproliferative responses to mitogens without alteration in cytotoxicity for tumor cells or susceptibility to bacterial or tumor cell challenge in mice exposed to TCDD has been observed by other researchers as well. Decreased antibody plaque responses with no effect on macrophage or NK cell function in TCDD-treated mice have been observed. These results are consistent with an increased susceptibility of TCDD-exposed mice to infection with influenza virus and a lack of effect on a Listeria bacterial challenge. TCDD, and other dioxin isomers, may also suppress serum complement levels in mice, resulting in an increased susceptibility to challenge with Streptococcus pneumoniae infection in these animals.

Exposure to TCDD during thymic organogenesis in rodents has resulted in more severe CMI suppression than that occurring following adult exposure. In some species, in utero exposure via maternal dosing appears to be necessary to induce maximum immunosuppression. At higher dosages, antibody responses and bone marrow stem cell numbers are depressed in most species. Administration of TCDD in utero also results in decreased resistance of offspring to bacterial and tumor cell challenge which correlates with altered CMI in these mice.

PCBs (CAS 1336-36-3). PCBs comprise a physicochemically and toxicologically diverse group of 209 compounds whose widespread use and chemical stability have made them ubiquitous in the environment. The chemical properties of commercial PCB mixtures depend on their degree of chlorination. PCB congeners with five to seven chlorine atoms per molecule are the most abundant in environmental matrices and the most bioaccumulative, while those that are more highly chlorinated are more tightly bound to particles and hence less bioavailable (reviewed by McFarland and Clarke, 1989; Safe, 1992).

Because of their generally low acute toxicity, effects on environmental receptors are more likely to be sublethal and chronic than acute. Toxicity and risk assessment of PCB mixtures is complicated by the fact that the 209 congeners differ markedly in both the severity and the nature of their biological effects. The lack of definition of PCB congeners present at INEEL is thus a major source of uncertainty in the WAG 9 ERA. The toxic potency of individual congeners is dependent upon their structure. While the approximate isostereomers of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)—i.e., coplanar molecules with chlorine atoms in the lateral (but not ortho) ring positions—are the most toxic (and carcinogenic in some species), many others manifest very low acute or chronic toxicity.

The most toxic congeners are also the most potent inducers of mixed-function oxidases as well as some Phase II enzyme activities (reviewed by Safe, 1992). These enzymes metabolize not only the inducing PCBs but also a variety of endogenous molecules, such as steroid hormones, that are necessary for normal physiological function. As a result, PCBs may exert adverse effects on development and reproduction in various vertebrate species, including birds (e.g., Koval et al., 1987). In addition, there is

considerable difference in the sensitivity of various species to these compounds. Particularly sensitive species include some birds, guinea pigs, and mink (McConnell, 1985).

Dahlgren and Linder (1971) and Dahlgren et al., (1972) examined the effects of Aroclor-1254 exposure in pheasants. Although no NOAEL was identified in this work, its focus on a wild species and dosing of both sexes makes it attractive for TRV development. A resultant TRV was developed for avian herbivores. Nine to 10 mg/kg-day Aroclor-1254 reduced sperm concentrations in American kestrels, *Falco sparverius* (Bird et al., 1983). A TRV for falconiformes was derived from this study. These TRVs are specific for the Aroclors (PCB mixtures) used in the source studies.

Linder et al., (1974) identified NOAELs for Aroclor-1254 in a two-generation reproductive study in rats. The TRV for small mammals was developed from this work. Many studies have focused on the toxicity of various PCBs to mink, which is a sensitive species (Eisler, 1986b; EPA, 1993). Related species such as otter and ferret are considerably less susceptible, suggesting that extrapolation from mink to receptors at INEEL may not be appropriate. However, in the absence of data for other mammalian carnivores/omnivores, a TRV for these species was derived from the work of Hornshaw et al., (1983).

For terrestrial plants, the recommended toxicological benchmark identified for PCB exposure was used as the TRV (Suter et al., 1993). No information on the toxicological effects of PCBs on reptile or invertebrate receptors was located.

6.3.6 Development of TRVs for Radionuclides

For radiological contaminants, injury is caused by absorption of energy in living tissue from the decay of radionuclides. As in the case of chemical toxicants, the dose of radiation absorbed by any individual organism is a function of its anatomy, physiology, ecology, and behavior.

Studies on the effects of radionuclides have shown that the rate of chronic exposure is more important than the total dose in assessing radiotoxicity (IAEA, 1992). The TRV values for all radionuclides and all animal functional groups was 1 mGy/day, which is the chronic dose below which there does not appear to be changes observed in terrestrial animal populations (IAEA, 1992). For terrestrial plants, the equivalent dose was 10 mGy/day, and this value was applied as the TRV for terrestrial plants for all radionuclides. Other available information on the effects of radionuclides on various functional groups is described below.

A chronic NOEL of 6 mGy/day was established for passerine birds based on no effect on breeding success of swallows and wrens exposed to 0.7–6 mGy/day (IAEA, 1992). The more conservative TRV of 1 mGy/day for avian groups was identified as described above in the IAEA (1992) report.

Redback vole populations (*Clethrionomys gapperi*) were unaffected at dose rates of 15 mGy/hour (IAEA, 1992). Chronic exposure of pigs and donkeys to 1 mGy/day was found to be an NOEL (Garner and Barber, 1966, as sited in IAEA, 1992). Chronic exposure of 4 mGy/day produced measurable declines in the number, motility, and viability of sperm in the dog, while exposure rates of <1.2 mGy/day failed to produce sperm count changes in dogs (IAEA, 1992). The more conservative TRV for mammalian groups is 1 mGy/day, which was identified for all animal functional groups (IAEA, 1992).

No significant differences in sex ratios, age distributions, or life spans were observed between lizards exposed to 20 mGy/day for 5 years and control iguanid lizards (IAEA, 1992). Conflicting results occurred when females of two other lizard species occupying the same enclosures became sterile, where reproduction

was blocked after 1 or 2 years, and the populations later drifted towards extinction. IAEA (1992) suggests that the ovaries of the two affected species (which live longer and mature slower than the iguanid lizards) would accumulate a greater total dose before sexual maturation. As identified above, the more conservative TRV for all animal functional groups is 1 mGy/day from the IAEA (1992) report.

The IAEA (1992) concluded that invertebrates appear to be more affected by indirect than direct effects. The direct effects of radiation on terrestrial invertebrates, insects in specific, are most likely limited by responses in fertility rather than mortality.

Doses of less than 10 mGy/day were assumed to represent an NOEL for plant species (IAEA, 1992). A more conservative TRV for all terrestrial plants is 10 mGy/day from the IAEA (1992) report.

6.3.7 Identifying Uncertainty Associated with TRVs

Although QCEs should be derived from the best available literature and all the uncertainties that could be reasonably accounted for are included in the AFs used to calculate TRVs, it is unlikely that any single scheme could suffice to extrapolate available toxicity data for all chemicals among all species. Thus, the remaining uncertainty in these criteria may be even greater than that associated with exposure estimation. Some of the extrapolations required in TRV development are listed in Table 6-19. TRVs are themselves dependent not only on extrapolation procedures but also on sampling adequacy and analytic accuracy, and the completeness and accuracy of response measurements in variable populations of test organisms. Combining results from different species, gathered under different experimental conditions, and extrapolation of results in test organisms to populations of resident species introduce additional, potentially significant sources of error as follows:

- While classical human toxicology relies on extrapolation of toxicity data from a handful of mammalian species to one species, an ecotoxicological evaluation must rely on extrapolation from a few test species to a larger number of receptor species spanning variable (and often large) ranges of phylogeny, anatomy, physiology, and life histories. Further, the spatial and temporal heterogeneity of exposure and conditions in natural systems can cause large variations in the doses and responses observed.

- Organisms in the environment are rarely (if ever) exposed to pure compounds alone, but rather to complex mixtures of chemicals for which the effects in combination are unknown.

- Chemicals may be volatilized, and transformed to more or less toxic products sequestered in the environment.

Our lack of knowledge of environmental variables and limited ability to replicate them in the laboratory or control them in the field results in a high level of uncertainty in our predictions of the effects of stressors on any given ecosystem component from laboratory toxicity tests.

6.4 Risk Characterization

Risk characterization is the final step of the WAG ERA process. The risk evaluation summaries the indication of risk due to the contaminant concentrations and exposure parameter-calculated dose for INEEL functional group, T/E, and C2 species and discusses the uncertainty inherent in the assessment. For a

Table 6-19. Extrapolations required for developing TRVs.[a]

Extrapolation	Example
Between taxonomic groups	From laboratory mouse to field mouse
Between responses to stressor	From mortality in dogs to a no-observed-effect-level in bobcats
Between laboratory and field conditions	From cage to steppe
Between individual animals to population	From decreased growth rate in captive individuals to effects on a wild population
Between short- and long-term exposure conditions	From acute or subchronic toxicity tests to lifetime exposure
Between laboratory and natural exposure media	Percent uptake of chemical mixed with laboratory diet vs. adsorbed to soil
Between spatial scales	Evaluation of the impact of exposure to a contaminated field on predators whose foraging range is 50 times as large

a. Adapted from EPA (1992).

WAG ERA, the evaluation step has two components starting with a description of the estimation of risk. A summary of the risk evaluation follows the risk estimation. These two components are described in the following sections.

6.4.1 Risk Estimation

An estimation of risk is made by comparing the calculated dose to TRV. Exposure parameters used to calculate dose to functional group, T/E, and C2 species are outlined in Section 6.2. Soil concentration data calculated in the human health risk assessment were used to calculate dose to ecological receptors at each site. The results of the dose calculations are presented in Appendix I. The use of human health concentration data is assumed to be more representative of the range of concentrations to which ecological receptors using a site at WAG 9 are likely to be exposed. If the dose from the contaminant does not exceed its TRV (i.e., HQs are less than 1 for nonradiological contaminants and 0.1 for radiological contaminants), adverse effects from exposure to that contaminant by ecological receptors are not expected, and no further evaluation of that contaminant is required. Hence, the HQ is an indicator of potential risk. TRVs are developed in Appendix G and discussed in Section 6.3.4. HQs are calculated using the following equation:

$$HQ = \frac{Dose}{TRV} \tag{6-32}$$

where

HQ = hazard quotient (unitless)

Dose = dose from all media (mg/kg-day or pCi/g-day)

TRV = TRV (mg/kg-day or pCi/g-day).

HQs are derived for all contaminants, functional groups, T/E, and C2 species identified in WAG 9 for each site of concern. The HQs from the results of the risk analysis are presented in Appendix I. If information was not available to derive a TRV then an HQ could not be developed for that particular contaminant and functional group or sensitive species combination.

An HQ greater than the target value indicates that exposure to a given contaminant (at the concentrations and for the duration and frequencies of exposure estimated in the exposure assessment) may cause adverse health effects in exposed populations. However, the level of concern associated with exposure may not increase linearly as HQ values exceed the target value. This means that the HQ values can not be used to represent a probability or a percentage, since an HQ of 10 does not necessarily indicate that adverse effects are 10 times more likely to occur than an HQ of 1. It is only possible to infer that the greater the HQ the greater the concern about potential adverse effects to ecological receptors.

6.4.2 Uncertainty Association With Hazard Quotients

For the WAG 9 ERA a HQ is used as an indicator of risk. The HQ is a ratio of the calculated dose for a receptor from a COPCs to the TRV. These ratios provide a quantitative index of risk to defined functional groups or individual receptors under assumed exposure conditions. The ratio, or HQ method is commonly used in both human health and ecological risk assessments. It is used in WAG ERAs to eliminate contaminants and sites that do not pose a risk to the ecosystem from further assessment.

In general, the significance of exceeding a target HQ (Table 6-12) value depends on the perceived "value" (ecological, social, or political) of the receptor, the nature of the endpoint measured, and the degree of uncertainty associated with the process as a whole. Therefore the decision to take no further action, consider corrective action, or perform additional assessment should be approached on a site-, chemical-, and species-specific basis. Because the unit of concern in ecological risk assessment is usually the population as opposed to the individual (EPA, 1992), exceeding conservative screening criteria does not necessarily mean that significant adverse effects are likely.

An HQ less than the target value (traditionally 1.0 for non-radionuclide contaminants) implies "low likelihood" of adverse effects from that contaminant. Nonradiological and radiological contaminants are treated separately because these two classes of contaminants cause different effects in exposed receptors.

The effects from the nonradioactive metals are expected to cause systemic toxicity, while the effects to reproductive processes are typically associated with exposure to ionizing radiation. A separate approach in which the target HQ is set to 1/n, where n is the number of nonradiological or radiological contaminants of concern, could also be used, while the HQ for could be set at 0.1 (1/10) for the radiological contaminants. This approach would be too conservative for nonradiological contaminants since it assumes cumulative (simultaneous) exposure to all nonradionuclides and that all contaminants within a given group behave synergistically in a given receptor. Given that all receptors within a functional group may not be simultaneously exposed to all contaminants, and that a synergistic effect may not be seen, this approach may be more stringent than necessary to protect all ecological receptors from nonradiological effects. Therefore, the HQ is set to 1 for all nonradiological contaminants. This method may underestimate the risk in that it does not account for cumulative exposure to multiple contaminants by a given receptor.

At this level in the ERA approach at the INEEL, both exposure and toxicity assumptions are generally "worst-case," and represent the upper bound of potential risks to ecological receptors. The HQ approach does not consider variability and uncertainty in either exposure or toxicity estimates, and therefore does not represent a statistical probability of occurrence of adverse ecological effects. Hazard quotients provide essentially a "yes or no" determination of risk and are therefore well-suited for screening-level assessments (EPA, 1988b). A limitation of the quotient method is that it does not predict the degree of risk or magnitude of effects associated with specified levels of contamination (EPA, 1988b). However, "modified quotient methods" are available that attempt to address this issue. For example, in the study of toxicity in fish, a method is used (Barnthouse et al. 1986) in which the conclusions are expressed as "no concern," "possible concern," and "high concern," depending on the ratio of the contaminant concentration to the reference (Barnthouse et al. 1986).

A summary of the WAG 9 ERA results is provided in Table 6-20. This table shows the order of magnitude for the largest observed HQ across all functional groups within the site vary by at least three orders of magnitude. The raw HQ results are shown in Appendix I.

6.4.3 Risk Evaluation

This section describes the results of the evaluation of risk associated with exposure of the functional groups, T/E, and C2 species to contaminants. The initial screening eliminated ten organic contaminants, four metals, and seven radionuclides. This resulted in one site being eliminated from the assessment (ANL-2). A total of 9 sites subsequently were assessed in this ERA. Generally, sites that have HQs greater than 1.0 are mainly due to metal contamination. Metals that appear to present the greatest potential for adverse effects include aluminum (HQs > 100 and < 1000), barium (HQs > 100 and < 1000), chromium(IV) (HQs >100 and <1000), copper (HQs > 100 and < 1000), cyanide (HQs > 100 and < 1000), lead (HQs >100 and <1000), magnesium (HQs >1000 and <10000), mercury (HQs >100 and <1000), sulfate (HQs > 100 and < 1000), vanadium (HQs > 100 and < 1000), and zinc (HQs >100 and <1000). There are three sites that have an HQ greater than 1.0 due to organic contamination; ANL-05 and ANL-35 because of dioxins/furans (HQs > 100 and < 10000), and ANL-61A due to PCB contamination (HQs > 1). There were no sites that have an HQ greater than 0.1 due to radionuclide contamination.

6.4.4 Discussion of Uncertainty

Uncertainty is inherent in the risk process and has been discussed in detail throughout this document. Principal sources of uncertainty lie within the development of an exposure assessment. Uncertainties inherent in the exposure assessment are associated with estimation of receptor ingestion rates, selection of acceptable HQs, estimation of site usage, and estimation of plant uptake factors and bioaccumulation factors. Additional uncertainties are associated with the depiction of site characteristics, the determination of the nature and extent of contamination, and the derivation of TRVs. All of these uncertainties likely influence risk estimates. Table 6-21 reviews the major sources and effects of uncertainties in the ERA.

6.4.5 WAG 9 ERA Summary

The objectives of this assessment were to define the extent of contamination for each site at the WAG level, determine the potential effects from contaminants on environmental receptors, habitats, or special environments, and determine the potential effects from contaminants on other ecological receptors at the WAG 9. The approach is an extension of the screening level ecological risk assessment methodology used at the INEEL (VanHorn et al., 1995). This methodology uses conservative exposure modeling and input parameters to identify contaminants and sites that may pose a risk to the environment.

Table 6-20. Summary of WAG 9 ERA results.

	Nonradionuclide		Radionuclide Internal		Radionuclide External	
Site	Contaminant	Hq[a]	Contaminant	HQ	Contaminant	HQ
ANL-01	Aluminum	>1000 and <10000	Initially Screened		Initially Screened	
	Antimony	>1 and <10				
	Asenic	>10 and <100				
	Barium	>10000				
	Bryllium	>1 and <10				
	Cadmium	>1000 and <10000				
	Chloride	>1 and <10				
	Chromium III	>1000 and <10000				
	Chromium VI	>100 and <1000				
	Copper	>10 and <100				
	Cyanide	>10 and <100				
	Lead	>10 and <100				
	Magnesium	>10000				
	Manganese	>10 and <100				
	Mercury	>100 and <1000				
	Nickel	>10 and <100				
	Selenium	>10 and <100				
	Silver	>10 and <100				
	Sodium	>1 and <10				
	Sulfate	>100 and <1000				
	Thallium	>1 and <10				
	Vanadium	>100 and <1000				
	Zinc	>100 and <1000				

Table 2-20. (continued).

	Nonradionuclide		Radionuclide Internal		Radionuclide External	
Site	Contaminant	Hq[a]	Contaminant	HQ	Contaminant	HQ
ANL-01A	Antimony	>1 and <10	Initially Screened		Initially Screened	
	Arsenic	>10 and <100				
	Barium	>1000 and <10000				
	Chromium III	>10 and <100				
	Chromium VI	>1 and <10				
	Cobalt	>1 and <10				
	Copper	>10 and <100				
	Cyanide	>10 and <100				
	Lead	>100 and <1000				
	Manganese	>10 and <100				
	Mercury	>100 and < 1000				
	Nickel	>100 and <1000				
	Selenium	>1 and <10				
	Silver	>1 and <10				
	Sodium	>1 and <10				
	Vanadium	>100 and <1000				
	Zinc	>10 and <100				
ANL-04	Aluminum	>1000 and <10000	Initially Screened		Initially Screened	
	Antimony	>1 and <10				
	Arsenic	>10 and <100				
	Barium	>1000 and <10000				
	Chromium III	>10 and <100				
	Copper	>100 and <1000				
	Cyanide	>100 and < 1000				
	Lead	>100 and < 1000				
	Magnesium	>10 and < 100				
	Mercury	>100 and <1000				
	Selenium	>10 and <100				
	Silver	>10 and <100				
	Sodium	>10 and <100				
	Vanadium	>100 and <1000				
	Zinc	>100 and <1000				
ANL-05	Sodium	>10 and <100	Initially Screened		Initially Screened	
ANL-09	Arsenic	>10 and <100	Initially Screened		Initially Screened	
	Copper	>1 and <10				
	Lead	>10 and <100				
	Mercury	>10 and <100				

Table 2-20. (continued).

	Nonradionuclide		Radionuclide Internal		Radionuclide External	
Site	Contaminant	Hq[a]	Contaminant	HQ	Contaminant	HQ
ANL-29	Silver	>10 and <100	None		None	
ANL-35	Aluminum	>1000 and <10000	Initially Screened		Initially Screened	
	Arsenic	>10 and <100				
	Barium	>1000 and <10000				
	Beryllium	>1 and <10				
	Cadmium	>100 and >1000				
	Chromium III	>1 and <10				
	Copper	>10 and <100				
	Cyanide	>10 and <100				
	HpCDD	>1 and < 10				
	Lead	>10 and <100				
	Magnesium	>1000 and <10000				
	Manganese	>100 and <1000				
	Mercury	>10 and <100				
	Nickel	>10 and <100				
	OCDD	>1 and <10				
	Selenium	>1 and <10				
	Silver	>100 and <1000				
	Sodium	>1 and <10				
	Sulfate	>1 and <10				
	Thallium	>1 and <10				
	Vanadium	>100 and <1000				
	Zinc	>10 and <100				
ANL-36	Silver	>1 and <10	None		None	
ANL-61A	None exceeded target value		None		None	

a. This represents the maximum HQs calculated across functional groups and T/E species.

Table 6-21. Sources and effects of uncertainties in the ecological risk assessment.

Uncertainty factor	Effect of Uncertainty (level of magnitude)	Comment
Estimation of ingestion rates (soil, water, and food)	May overestimate or underestimate risk (moderate)	Few intake (ingestion estimates used for terrestrial receptors are based on data in the scientific literature (preferably site-specific) when available. Food ingestion rates are calculated by using allometric equations available in the literature (Nagy 1987). Soil ingestion values are generally taken from Beyer et al. (1994).
Estimation of concentration and plant uptake factors	May overestimate or underestimate risk and the magnitude of error cannot be quantified (high).	Few bioaccumulation factors (CFs) or plant uptake factors (PUFs) are available in the literature because they must be both contaminant- and receptor-specific. In the absence of more specific information, PUFs and CF/s for metals and elements are obtained from Baes et al. (1984), and for organics from Travis and Arms (1988).
Estimation of toxicity reference values	May overestimate (high) or underestimate (moderate) risk	To compensate for potential uncertainties in the exposure assessment, various adjustment factors are incorporated to extrapolate toxicity from the test organism to other species.
Use of functional grouping	May overestimate (moderate)	Functional groups were designed as an assessment tool that would ensure that the ERA would address all species potentially present at the facility. A hypothetical species is developed using input values to the exposure assessment that represents the greatest exposure of the combined functional group members.
Site use factor	May overestimate (high) or underestimate (low) risk	Site use factor is a percentage of the site of concern area compared to home range of the receptor species. Home range is not known for many species and therefore a default of 1.0 is used. This can overestimate the risk at small sites.

The WAG 9 ERA incorporates levels of uncertainty that could either overestimate or underestimate the actual risk to these receptors. To compensate for potential uncertainties, the WAG 9 ERA incorporates various adjustment factors that are designed to be conservative rather than result in a conclusion of no indication of risk when actual risk may exist. Regardless of the inclusion of adjustment factors, other uncertainties exist that could affect the estimation of true risk associated with WAG 9.

The basis of the TRVs developed for non-radionuclides is effect to the individual. This conservative approach is very commonly used due to the large uncertainty inherent in extrapolating effects data from test to field organisms. Conservatism is also compounded by the limited level of exposure modeling (i.e. transport of contaminants in the food chain from the subsurface to surface). Using this level of analysis and given that individual ecological receptors are presented greater exposure than human occupational scenarios all nine sites exhibited risk to ecological receptors from non-radionuclides.

The primary value of the WAG 9 ERA is to identify COPCs which exceed the ecological hazard quotients for the various sites. The sites with high ecological hazard quotients will be retained and evaluated in the feasibility study. The feasibility study will determine which sites pose an unacceptable ecological risk. Those sites with unacceptable ecological risks will be remediated during the remedial action for WAG 9.

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